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PART I.

ORIGINAL COMMUNICATIONS.

ART. I.—*An Experimental Inquiry into the Pathology and Treatment of Asphyxia.* By JOHN E. ERICHSEN, Lecturer on General Anatomy and Physiology at the Westminster Hospital, London.

HAVING had the honor of being appointed by the General Committee of the British Association for the Advancement of Science, at the meeting at Manchester in the year 1842, one of a commission, consisting of Dr Sharpey and myself, to make an Experimental Inquiry into the subject of Asphyxia, I have much pleasure in laying the following Report before the Association. This would no doubt have been much more deserving of the attention of the members of this institution than it at present is, had not the pressure of other engagements prevented my most distinguished friend and coadjutor, Professor Sharpey, from continuing conjointly with me the investigation of this interesting but very intricate subject. It would be useless to recapitulate the precise share that each of us took in the inquiry; but I may state in general terms, that in several of the experiments that it was necessary to have recourse to, I had, more particularly during the earlier part of this investigation, the great advantage of that gentleman's guidance and most valuable co-operation; that others were performed altogether by myself; and that I alone am answerable for this Report, and for the opinions maintained therein.

The principal points to which my attention has been directed in drawing up this Report, have been the cause of the cessation of the circulation in, and the treatment of, asphyxia.

There are few subjects in the whole range of the medical sciences about which more controversy has arisen, in which many of the most distinguished physiologists have taken an active part, than the immediate cause of death, and the order of the cessation of the vital actions in asphyxia. This is not to be wondered at when we reflect upon the intimate connection that exists between the respiration, the heart's action, and the functions of the nervous centres in man and the higher classes of animals; and how beset with innumerable difficulties, and how liable to error any experimental inquiry must be in which we endeavour to study the pathology of functions, individually and singly, that are naturally closely linked together, and intimately dependent upon one another. As Dr Carpenter very justly observes, "When any link in the chain of the vital phenomena is broken, and the equilibrium of the whole disturbed, the derangements that ensue are so various and complicated that it is exceedingly difficult to assign to each its peculiar agency in finally producing the fatal termination." When we consider that the subject of asphyxia has engaged the attention of physiologists since the time of Haller, the progress that has been made in it has been but very slow, each succeeding inquirer having only gradually cleared away those sources of fallacy that caused a wrong explanation to be given of the facts that had been established by his predecessors. And it is only by insulating carefully the different phenomena that present themselves, and by studying them singly, or, if this cannot be accomplished with due attention to those modifying circumstances that it is impossible to remove, that a correct knowledge can be obtained of this complicated subject. We will first proceed to examine in detail the principal doctrines that have at various times been promulgated respecting the cessation of the circulation in asphyxia.

These may be arranged under the three following heads:—*

1*st*, That the circulation ceases in consequence of the arrest of the respiratory movements.

2*d*, In consequence of want of power in the heart.

3*d*, In consequence of an obstruction in the passage of the blood through the capillaries of the lungs.

1. The doctrine, that the cessation of the circulation was due to the arrest of the respiratory movements, was entertained by the great Haller, who supposed that the pulmonic capillaries being

* I would refer those who would wish to pursue the history of this subject more in detail, to Dr Kay's very valuable Treatise on Asphyxia, or to the very lucid article by Dr Carpenter on the same subject in the third volume of the Library of Practical Medicine.

compressed by the collapse of the lungs, the circulation through them was arrested. This opinion was disputed by Goodwyn, who proved that the circulation continues through a lung that is compressed by the effusion of fluid into the chest. It may also be shown to be, at all events, not the sole agent in the cessation of the circulation, by the fact of an animal being readily asphyxiated when made to breathe nitrogen, or whose lungs are kept distended with vitiated air.

It must be borne in mind, however, that these experiments merely prove that the arrest of the respiratory movements is not the only cause in bringing the circulation to a stand, and not that it is without any influence in this respect.

But although Haller's doctrine is not of itself sufficient to account for the arrest of the circulation, yet I am inclined to think that the cessation of the respiratory movements have, independently of any other cause, some influence, although perhaps not a very powerful one, in occasioning this phenomenon. I am the more inclined to this opinion, as I have frequently observed that when a pulmonary vein is punctured, whilst artificial respiration is being kept up, the flow of blood from the puncture is evidently increased during the distension of the lung; and indeed, when the circulation has become much enfeebled, it ceases entirely during the collapse of that organ.

The experiments by which Dr Alison endeavours to prove that the stagnation of the blood in the lungs is due solely to the cessation of the changes between it and the atmospheric air in the pulmonic capillaries, and not to the arrest of the mechanical movements of the chest, do not appear to me to be altogether conclusive. This eminent physiologist confined a rabbit in nitrogen gas until its respiratory movements became laboured and insensibility was approaching. The animal was then withdrawn as rapidly as possible, and the brain having been suddenly crushed by the blow of a hammer, the chest was laid open. As the quantity of blood found in the right side of the heart preponderated considerably over that in the left, and as the respiratory movements had not been interrupted until the animal was deprived of life, this experiment was looked upon as conclusive evidence of the fact, that the accumulation of the blood in the right side of the heart was owing, not to the arrest of these movements, but to a cessation of the chemical changes between the blood and the air.

This conclusion appears to me not to be altogether warranted by the facts of the case; for it must be remembered that, in this experiment, the circulation continues after the cessation of the respiratory movements. When an animal that has been breathing atmospheric air up to the moment of its death, is killed by a violent blow upon the head, the sensorial functions are im-

mediately annihilated and the respiratory movements are arrested; but the heart continues still to beat, and black blood to circulate, as in ordinary cases of asphyxia, for some time after the extinction of animal life; the cessation of the action of that organ only ensuing in consequence of the diminution in the quantity and the alteration in the quality of the blood with which it is supplied; and after death the ordinary appearances of asphyxia present themselves, though the chemical changes have continued as long as the respiratory movements. The following is, according to Bichat, the order of the cessation of the vital actions in severe injuries of the brain, as when that organ is crushed:—1. Interruption of the cerebral action; 2. Annihilation of the action of all the muscles of animal life; 3. Consequent cessation of the respiratory movements; 4. Suspension of the chemical changes, and consequently of the coloration of the blood; 5. Permeation of fibres of the heart with black blood; 6. Weakening and cessation of the action of these fibres.* Now, in Dr Alison's experiment, supposing that the chest were laid open and the heart exposed as rapidly as possible after the death of the animal, at the very least a minute or a minute and a half must have elapsed, after the cessation of the respiratory movements, before this could be accomplished, during which the heart would be acting strongly; and sufficient time would elapse for the circulation to make at least one or two circuits. Blood would consequently, as in all cases of asphyxia, from whatever cause, accumulate in the right cavities of the heart.

As an accumulation of blood in these cavities is therefore a necessary result of the sudden annihilation of the functions of the brain, in consequence of the suspension of the respiratory movements or of the chemical changes in the lungs—one or both—when the animal has, up to the moment of its death, been breathing atmospheric air, the same phenomenon ought not, I think, to be adduced as an effect of the cessation of the chemical changes alone, where it is met with in an animal that is killed whilst breathing nitrogen,—there being no evidence that it existed before the death of the animal, and that it did not result from the continuance of the circulation after the cessation of the respiratory movements.

It was thought that the relative influence of the arrest of the respiratory movements, and of the cessation of the chemical changes in occasioning an accumulation of blood in the cavities of the heart, might be more accurately determined by keeping up the movements of respiration, not only until after these changes had been arrested, but until the pulsations of the heart and the circulation of the blood had entirely ceased. The difference in the

* Recherches Physiologiques, p. 320.

proportional quantity of blood contained in the two sides of the heart in this, and in ordinary cases of asphyxia, being the measure of the relative influence of the cessation of the chemical changes, and of the arrest of the respiratory movements. The following experiment was accordingly performed :—

Exp. 1. A tube having been introduced into the trachea of a small black terrier, a syringe capable of holding eight ounces, and filled with air, was attached to it. The piston was then worked in such a way that the natural movements of respiration might be imitated.

$1\frac{1}{2}$ *minute.*—Animal began to struggle.

3d minute.—All struggles ceased.

11*th minute.*—The chest was laid open, and the lungs found fully distended and of a darkish gray colour.

14*th minute.*—Heart's action ceased entirely. During the whole of this time the action of the syringe had been continued except whilst the chest was being laid open, which renders the experiment perhaps a little less conclusive than it otherwise would have been.

The heart was found much distended on both sides ; coronary arteries and veins quite black ; the pulmonary veins as well as artery, were fully distended and perfectly black. The left auricle being punctured, a large quantity of fluid black blood flowed from it and from the pulmonary veins ; the left ventricle also contained a considerable quantity of black fluid blood ; the right cavities contained a large quantity. The difference, however, in the quantities contained in the opposite sides of the heart was not by any means so great as usual ; the left side containing comparatively much more blood than in ordinary cases of asphyxia ; indeed, very nearly as much as was found in the right cavities. This experiment I have repeated several times with the same result, the quantity of the blood in the left cavities being invariably greater than in those cases of asphyxia in which the respiratory movements have, from the first, been arrested. This is in accordance with the observations of Kite and of Coleman, who found that when the lungs were kept mechanically distended during the process of asphyxia, the left cavities of the heart contained comparatively more blood than the right ; in some cases the quantities in the two opposite sides of this organ differing very little, if at all.

From these experiments, then, I think that we may conclude that the persistence of the respiratory movements has some influence in maintaining the circulation through the lungs, but that their arrest is not by any means the sole cause of the cessation of the circulation.

2. We will now proceed to the consideration of the second class of opinions, namely, that the circulation ceases in asphyxia

in consequence of a want of power in the heart. This cause, which was much insisted upon by the earlier writers, appears to me to have had scarcely sufficient attention paid to it by several modern physiologists; more especially in estimating the influence of an obstructing cause in the capillaries on the cessation of the circulation.

It will be unnecessary to do more than allude to the opinion of Goodwyn, that the circulation was arrested and the heart's action brought to a stand in consequence of the venous blood being an insufficient stimulus to the left cavities of that organ, as this doctrine received a complete refutation from Bichat, who found that the injection of venous blood into the left cavities will stimulate them when their action is much depressed; and that blood of a perfectly venous character circulates for some time through the pulmonic and systemic capillaries before the heart's action ceases. In hybernating animals, also, blood that is nearly of a venous character circulates without difficulty. Besides, were Goodwyn's views correct, the left cavities of the heart and the pulmonary veins ought to be found after death distended with blood, which is not the case.

The opinions of Bichat on this part of our subject are of the highest interest and importance. This physiologist supposed, as is well known, that the cause of the cessation of the circulation in asphyxia was that venous blood penetrated into the tissue of the heart, weakening each individual fibre; and he states that he is convinced that if black blood could be made to circulate through the coronary artery, whilst arterial blood passes as usual through the cavities of the heart, that the action of that organ would cease as rapidly as when venous blood only penetrates into its tissue by the coronary arteries, after having passed through the left cavities of that organ. Thus, he says, it is only little by little, and after it has thoroughly penetrated into each individual fibre, that the action of this organ is lessened, and at last ceases; in the other case, (according to Goodwyn's view,) a sudden cessation of the heart's action ought to ensue. It would appear that Bichat did not believe that venous blood was actually deleterious to muscular contractility, but rather that it acted by the exclusion of arterial blood. He says, "le cœur cesse d'agir lorsque les phenomenes chimiques du poumon sont interrompus parceque le sang noir qui penetre ses fibres charnues n'est pas propre à entretenir leur action." He did not, however, regard the enfeebled condition of the heart as the sole cause of the cessation of the circulation in asphyxia. This he attributed likewise to an increase in the mass of the blood, owing to a retention in it of excrementitious matters, which in a healthy condition pass off by the different secreting organs. He also thought that there was a tendency to the ob-

struction of the blood in its passage through the tissue of the lungs in consequence of a want of due stimulation of that organ by red blood; the black blood circulating through the bronchial arteries producing the same effect upon the lungs as it does when it penetrates the tissue of the heart. Another cause which embarrasses the circulation through the lungs, when the chemical changes cease, is according to Bichat, the absence of the stimulus of the vital air upon the mucous membrane of the air cells, by which he supposed the capillary circulation to be, in a normal condition of that organ, partly maintained; and lastly, as far as the heart is concerned, the cause that gives rise to the stagnation of the blood is the enfeebled condition of the right auricle and ventricle, which being permeated by the black blood, are no longer capable of impelling this fluid with sufficient energy towards the lungs, and consequently of overcoming the resistance that it meets with there.

I have been thus particular in giving a résumé of the opinions of Bichat on asphyxia, as they cannot, I conceive, be too attentively studied by any one who wishes to make himself acquainted with the pathology of this disease.

It is almost needless to adduce any formal experimental evidence in proof of the fact, that the force of the contractions of the heart is weakened in asphyxia; but it may not be altogether uninstructive to inquire how this debility is occasioned.

The influence of arterial blood in maintaining the contractility of the voluntary muscles has long been recognized both by physiologists and surgeons. Stenson, Emmert, Bichat, and Segalas, have proved by direct experiment upon animals, that on ligaturing the larger arterial trunks, the limbs from which the supply was thus cut off were found to be much weakened and indeed paralysed in their muscular power. It is also a fact familiar to all practical surgeons, that, after the ligature of the main artery of a limb, as the femoral or axillary, for instance, voluntary motion will be lost in the part which is thus deprived of its blood; and in these cases it is often many weeks before the power of moving the extremity is regained. That the same is the case in the heart I have already attempted to show by some experiments, the details of which were read before the British Association in 1842, and which were published in the Medical Gazette of the same year. From these it would appear, 1st, That the arrest of the coronary circulation produces a speedy cessation of the heart's action, though respiration be actively carried on; and, 2d, That the presence of venous blood in the fibres of the heart serves to maintain the contractions of that organ for a longer period than when its tissue is entirely deprived of blood. Dr Kay had previously, satisfactorily, and incontestably ascertained that the contractile power of muscles, involuntary as well as volun-

tary, is supported through a comparatively longer period when their fibres are congested even with venous blood, than when emptied of that fluid; but that, nevertheless, dark blood is less favourable than arterial to the contractility of muscle. Now in asphyxia we have both causes, a diminution in the quantity, and an alteration in the quality of the blood, in operation; the blood that reaches the left cavities, and that is consequently propelled into the coronary arteries, being, after a time, much diminished in quantity, and being also deprived of its normal stimulating qualities by its conversion into venous. Indeed, on referring to the very interesting experiments of Dr Reid, on the obstruction that takes place in the systemic capillaries in asphyxia, it will be found that the maximum of the pressure, and consequently of the force of the heart's contractions, is at about the second minute after the closure of the trachea, and that, after that, the pressure gradually falls, although the obstruction continues the same. This is in accordance with what I have likewise observed on repeating and modifying these experiments.

I cannot, however, agree with Dr Kay as to the length of time that the heart will continue to contract after the circulation has ceased. He says, "that it contracts long after blood has ceased to flow from the coronary veins;" and again, "its contractions survive the period when it is possible that the coronary arteries can receive the asphyxial blood."

I think that it may fairly be questioned whether this is the case; whether the contractions of the heart continue at all after the circulation through the coronary vessels has ceased. And not only through the coronary, but also through the pulmonary vessels. For it must be borne in mind, that the left cavities of the heart are never found empty in asphyxia; that although not distended, as the right auricle and ventricle are, yet that they are frequently in this disease found to be tolerably full of blood. Coleman states that the relative quantities of blood in the two sides of the heart varies considerably, but that on an average the quantity in the right is to that in the left as $3\frac{2}{8}$th to $1\frac{6}{8}$th, or as about $3\frac{2}{3}$ds to 2. Now such a large quantity of blood as this, amounting to nearly two-thirds of that in the right cavities, can certainly not be supposed to accumulate in the left side of the heart if the contractions of that organ continue long after the circulation has been brought to a stand. On the contrary, it would lead to the opposite inference, or, at all events, prove that the stoppage of the circulation could not have occurred before the cessation of the heart's action. And this I believe to be the case that the circulation is not entirely arrested until the heart has ceased to beat.

On taking the average of nearly twenty experiments on asphyxia, in which the time that the contractions of the heart continued

after the trachea had been closed was noted, I find that the contractions of the ventricles last for about nine and a quarter minutes, and that the pulsations in the femoral artery are perceptible on an average for about seven and a half minutes after all access of air to the lungs has been prevented. Now Bichat states that the blood in the arteries becomes as dark as that in the veins in from a minute and a half to two minutes, which statement is confirmed by Dr Reid, and the accuracy of which I have had frequent occasion to remark; the left ventricle will therefore continue to propel perfectly venous blood for at least six or seven minutes, with a sufficient degree of force to occasion a pulsation in the femoral artery, and *a fortiori* therefore into the coronary artery. Supposing that the circulation through this vessel ceases entirely when the pulse can no longer be felt in the femoral artery, the heart would only contract for at most two minutes after the passage of the blood through the coronary vessels had ceased. But it is extremely probable that although the contractions of the heart have become so feeble after about the seventh or eighth minute as not to be able to propel blood into the femoral artery, yet that they may still continue to do so in a small and enfeebled stream certainly, but still to a certain extent into the coronary arteries; from which I have never failed to procure blood by puncture as long as the heart's action continues. It must, moreover, be borne in mind that there is never a complete obstruction in the capillaries of the lungs, a certain quantity of blood finding its way into the left cavities of the heart until the very last, as these are never found perfectly empty after death by asphyxia, but most generally, indeed, contain a tolerably large quantity of blood. So that the blood is present, provided the contractions of the heart are not so excessively enfeebled as to be unable to propel it into the coronary arteries, which cannot require much force; indeed, if the blood be merely ejected, or trickle out of the ventricle, it will find its way into these vessels, as is proved by the fact, that when artificial respiration is set up after all ventricular action has ceased, the coronary arteries, which had previously been quite black, are seen to acquire a florid hue, without any ventricular contractions having taken place, but solely in consequence of the draining of a certain quantity of the aërated blood into these vessels from the left ventricle, showing with what ease nature intends the blood to pass into the coronary arteries.

Another very interesting and important fact connected with the action of the heart in asphyxia is the great diminution in the frequency of its pulsations, as soon as the blood in the arteries has become perfectly black. This fact has been noticed by Dr Reid, though in reference to another point in the pathology of the disease. There is less occasion, therefore, to give a detail of the results of the observations that I have made upon it, though it may

be as well, for the sake of illustration, to mention the details of two of the cases in which I noted them.

Exp. 2. The animal was asphyxiated by having the stop-cock of a pipe that had been fixed in his trachea shut, and the pulsations were noted on the femoral artery, which had previously been exposed, as in Dr Reid's cases. In the first experiment the heart was acting tumultuously, at least 130 in a minute; at the end of the first half minute after the stop-cock was turned it had fallen to 104—108; whilst the animal was struggling during the next two minutes it rose again in frequency, but could not be counted owing to its tumultuous action; at the end of the third minute it had fallen to 48–52, at which time the animal was perfectly quiet and quite insensible; it gradually fell, until, at the end of the fifth minute, it was 36–40, and rather irregular; at the sixth minute it had fallen to 28; at the seventh minute it was about the same, but much weaker and more irregular; and before the eighth minute it had ceased to be perceptible.

Exp. 3. In the next experiment the pulse was at the time of the closure of the stop-cock 100—104, pretty regular; at the end of the first minute, when the animal was struggling violently it had risen considerably, to at least 130 or 140, but could not be counted with certainty, owing to its tumultuous and irregular action; at the end of two and a half minutes it had fallen to 68–72, at which time the animal was perfectly quiet, and the blood as black as in the accompanying vein; at the end of the third minute it had fallen to 60–64. The pipe in the trachea was then opened, and on air being admitted to the lungs, it immediately rose to 76–80, showing the influence that the aëration of the blood has in immediately restoring the frequency of the heart's contractions. The effect that it has in restoring their power is evident from Dr Reid's experiments, in which the mercury rose from 6 to 11 inches when a bladder of atmospheric air was applied to the trachea.

These observations, which it is needless to multiply, prove that the frequency of the heart's contractions is diminished by from one-third to one-half when perfectly venous blood is circulating through its vessels. The rapidity of the circulation, and the propulsive force exercised by the heart during a given time, must therefore, *cæteris paribus*, be proportionately lessened.

It was thought that additional light might be thrown upon this part of our subject by observing the phenomena of asphyxia in those young mammals, which have the eyes closed for some time after birth, and in which the *foramen ovale* and *ductus arteriosus* are still open; it being evident that in these animals death could not occur from obstruction of the pulmonary circulation, the two sides of the heart communicating in another and more direct way than through the pulmonary vessels.

The very interesting experiments of Buffon, Legallois, and Edwards, on the period that young warm-blooded animals will live when deprived of air, are so well known as not to require more on our part than a passing reference. Legallois found that the power that some young mammals possess of enduring a deprivation of air rapidly diminishes as the age of the animal increases; and Edwards pointed out the fact, that the duration of life, when animals are deprived of air, is chiefly influenced by two circumstances, namely, their power of generating animal heat, and the external temperature to which they are exposed. In young puppies, shortly after birth, the quantity of blood that passes through the lungs, and that consequently becomes arterialized, whilst the *foramen ovale* and *ductus arteriosus* remain open, is but very small. This no doubt increases day by day until these passages are closed up, the vital condition of the animal becoming such as gradually to necessitate the arterialization of a larger quantity of blood. But yet limited though the quantity of arterialized blood, during the earlier period of existence, may be, yet the complete deprivation of that fluid is incompatible with life, the animal being as certainly, though not as quickly, destroyed on the exclusion of atmospheric air as an older one is.

Exp. 4. A puppy, four days old, was strangled. Struggles ceased in nine minutes. The thorax was then laid open, and the heart exposed; the blood that flowed from the cut mammary and intercostal arteries was perfectly black. At the expiration of an hour and twenty minutes the ventricles had ceased to act; the auricles continued to act for nearly three hours and a half.

On examination about two hours after the action of the heart had ceased, it was found that the right cavities and pulmonary artery were full of black blood, but by no means distended,—certainly not containing so much as in older animals that have been asphyxiated. The left cavities also contained a considerable quantity of black blood, but not quite so much as the right side did; there was likewise some in the aorta. The *ductus arteriosus* and *foramen ovale* were quite pervious. The lungs were apparently but slightly congested.

Exp. 5. Another puppy of the same age was strangled; the spasmodic movements continued for sixteen minutes; the chest was examined four hours after the trachea had been tied. All movement of the heart had then ceased, and the same appearances, as nearly as possible, were found as in the former case,—the difference in the quantity of blood on the two sides of the heart being but small. The same experiment repeated on other puppies of the same age was attended with similar results.

In the asphyxia of very young animals it is quite evident, as has already been stated, that the circulation cannot be brought to

a stand in consequence of any obstruction to the passage of the blood through the pulmonary capillaries, as the *ductus arteriosus* and *foramen ovale* are still patent. This is also proved by the fact of both sides of the heart containing nearly an equal quantity of black blood. The cause of death must consequently be sought for in the heart; which organ continues acting until its irritability, which (like that of the muscular system generally in young warm-blooded animals with closed eyes) is very great, is exhausted by the continued circulation of black blood through its muscular fibres. For however low the vitality of a young puppy may be, yet the process of respiration is carried on to a certain extent, it being necessary, as has already been stated, for the maintenance of life, that a certain quantity of arterial blood should circulate through the system. When this supply, comparatively trifling as it may be, is cut off, the blood becoming entirely venous, is rendered unfit for the supply of the wants of the system; for although, as Edwards and Dr Kay have shown, venous blood may for a time keep up the contractility of the heart and other muscles, yet it is of course unable to do so for a continuance.

The principal cause of the cessation of the circulation, then, in young warm-blooded animals born with the eyes closed, whilst the *ductus arteriosus* and *foramen ovale* remain open, appears to be the gradual diminution in the force of the heart's contractions in consequence of the circulation of black blood through its muscular fibre.

The following are the phenomena, then, of the cessation of the heart's action in asphyxia: When the access of atmospheric air is prevented, the blood continues to be supplied for a minute or two, apparently in its usual quantity, by the pulmonary veins, and the contractions of the heart are as strong, and usually more frequent than natural. At about the second or third minute, when the blood has become perfectly venous in its characters, the frequency of the contractions falls very considerably, and the force of the propulsive power probably begins to be impaired. The alteration in the quality of the blood continuing, and the diminution in its quantity increasing, owing to causes that will presently be adverted to, the contractions of the ventricles become graudally weaker and weaker, as well as less frequent, and at about the ninth or tenth minute cease entirely; tremulous movements continuing in them, however, without distinct contractions, for some little time longer.

3. We now come to the consideration of the third class of opinions, viz. that the cessation of the circulation arises principally or entirely from an obstruction to the passage of the blood through the capillaries of the lungs. About the evidence of such an obstructive force there can no longer be any doubt; but the manner in which it is occasioned is still a *quæstio vexata* amongst physiologists.

That some degree of obstruction takes place in the capillaries of the lungs there can be no doubt. There is sufficient evidence of this in the congested condition of the lung, in the tension of the pulmonary artery, and in the accumulation of blood in the right cavities of the heart, as compared to the state of the pulmonary veins, and of the left auricle and ventricle.

The recent experiments of Dr Reid (Edin. Med. and Surg. Journ. Vol. lv.) have established the important fact, that the circulation is obstructed in the systemic as well as in the pulmonic capillaries, causing an increased pressure on the walls of the arteries, as ascertained by the hæmadynamometer, whilst a similar instrument, when introduced into a vein, exhibits a proportionate diminution of the pressure in that class of vessels. From this it is evident that in asphyxia there exists an occasional impediment to the passage of the blood from the arteries into the veins.

I must confess, that on perusing the results of Dr Reid's experiments, and on repeating these on animals, I was at first inclined to attribute this retardation in the passage of the blood through the systemic capillaries, simply to the effect of the compression of these vessels, and the consequent concentration of the heart's force in the arterial system, by the violent struggles and efforts at expiration of an animal that is being asphyxiated. This opinion was strengthened on looking over the table of the details of one of these experiments, in which it will be found that the greatest range in the mercury occurs whilst the animal is struggling violently. I have several times seen the mercury in a hæmadynamometer that has been fixed in the carotid artery of a dog, oscillate between four and eight inches if the animal struggled or expired forcibly, although the respiration was free and unimpeded. This objection appears to have occurred to Dr Reid, as he makes, in the very interesting paper referred to, some remarks on the increased force with which the blood is sent along the arteries during violent muscular contractions, and especially during expiration. In order to satisfy myself of the influence of this cause in Dr Reid's experiments, which is of some importance in general physiology, as well as in its bearings on the pathology of asphyxia, I determined to ascertain whether the pressure in the arteries is increased during the progress of asphyxia in an animal in which no muscular movements can take place; and if it were increased, to what extent. By ascertaining this, the pressure that is due to the muscular movements of the animal may be separated from that which is dependent upon an obstruction in the capillaries from some other cause. I accordingly performed the following experiment,

Exp. 6. A pipe with a stop-cock attached was introduced into the trachea, and a hæmadynamometer was adapted to the carotid

artery of a middle-sized dog. The animal was then pithed, and artificial respiration was immediately set up. The mercury in the hæmadynamometer stood at 3½ to 4 inches, and the heart was beating 80—84 in a minute. On discontinuing the artificial respiration, which was done as soon as the pressure in the arterial system had been ascertained, the mercury in the hæmadynamometer remained at the above level for about half a minute; it then began to rise, and by the end of the first minute stood at 5¾ to 6½ inches, at which point it continued for about two minutes, when it gradually began to fall in consequence of the failure of the heart's action, which ceased entirely at the ninth minute.

Exp. 7. In another experiment of the same kind, after the animal had been pithed and artificial respiration set up, the mercury in the hæmadynamometer stood at 3½ inches, oscillating between that point and 4½ inches. On discontinuing the inflation, which was done as soon as the pressure had been taken, the mercury remained stationary for about a quarter of a minute; it then gradually rose, and at the end of the first minute stood at 5½ and 6 inches, occasionally rising to 6½ and 6¾ inches; it stood at this height for a minute and three-quarters, when it gradually and slowly began to sink, and at the eighth minute, when the ventricles ceased to beat, stood at a level.

Exp. 8. I performed the same experiment a third time with a like result, the mercury rising steadily, soon after inflation was discontinued, for 2 or 2½ inches, and maintaining this level for about two minutes, when it gradually and slowly began to sink, in consequence of the failure of the heart's action, which gradually became weaker and weaker, and ceased entirely before the tenth minute. These experiments fully confirm those that have already been published by Dr Reid, and serve to establish the fact that the blood, when it has become venous, is retarded in its progress through the systemic vessels. They differ, however, from the results obtained by this gentleman in one important particular, namely, the much smaller rise in the mercury, and the consequently smaller amount of pressure in the arterial system, which I am at a loss to account for in any other way than by attributing it to the total absence of one cause of obstruction,—muscular contraction. It is true that the operation of pithing may, like the sudden destruction of the brain, weaken the force of the heart's action, and this may account in some measure for the absolute rise of the mercury in the hæmadynamometer not being in my experiments somewhat greater than it was, but it cannot be the sole cause; for the increase of pressure after the blood had become venous was, relatively to what it was whilst inflation was being performed, smaller in my experiments than in Dr Reid's; amounting only to from 2 to 2½ inches of mercury, or to about a

pound to a pound and a quarter on the square inch; which was maintained until the heart's action became enfeebled from the want of its proper stimulus, when the circulation was gradually brought to a stand throughout the body. The relative difference, then, in these two sets of experiments in the height of the mercury before and after the blood became venous, can only, I think, be accounted for by the absence in one of them of the obstructive influence of muscular contraction.

I think it probable that the retardation must occur in the pulmonic vessels before it does in the systemic ones, as we should otherwise find it difficult to account for the accumulation of blood that is invariably found to take place in the venous system in asphyxia. That the retardation of the blood should commence, and perhaps be more complete, in the pulmonic than in the systemic vessels, is nothing more than we should expect, as those vessels are more immediately and directly influenced in the process of asphyxia than those of any other organ or part of the body.

The next question, how this obstruction is occasioned, is the most important of all that present themselves in this inquiry, and its solution is a matter of some moment to physiological science.

There are at present three modes of accounting for this phenomenon, each of which can boast of distinguished supporters. They are as follows:—

1*st*, That the cessation of the circulation through the capillaries of the lungs is the result of a sort of paralysis of these vessels, occasioned by the deleterious action which the venous blood exercises on the nervous centres in its circulation through them, whereby the "nervous influence" that is necessary for the flow of blood through the lungs is destroyed.

2dly, That the stagnation of the blood in the lungs is the result of the cessation of the vital attractions connected with the chemical changes between the air and the blood during the process of respiration.

3dly, That, in consequence of the non-arterialization of the blood, the minute pulmonary vessels which usually convey arterial become incapable of transmitting venous blood, which consequently stagnates in the lungs.

a. We will first of all proceed to the consideration of the doctrine, that the cessation of the circulation in the capillaries of the lungs is the result of a sort of paralysis or loss of tone of these vessels, occasioned by the deleterious action which the venous blood exercises on the nervous centres by its circulation through them, whereby the "nervous influence" that is necessary to the flow of blood through the lungs is destroyed. That the non-arterialized blood flows freely through the vessels of the lungs into the left side of the heart, whence it is propelled into the general

circulation, until its deleterious influence has so enfeebled the actions of the nervous centres that the capillaries of the lungs become unable to transmit the blood that is propelled into them from the right side of the heart. Now it is certainly an incontestable fact that the actions of the brain and *medulla oblongata* become gradually more and more enfeebled during the progress of asphyxia, but that the cessation of the actions in these parts is the cause of the arrestment of the circulation through the lungs is not so evident.

It would be out of place here, and would lead us too far away from our present subject, to inquire into the evidence on which the doctrine of the dependence of the capillary circulation on nervous influence rests; but, as Dr Carpenter remarks, "The doctrine that nervous influence is essential to the flow of blood through the capillaries of the lungs or of any other part is a mere assumption unsupported by physiological facts." The principal objections that bear against this explanation are, that the circulation through the lungs may, as will hereafter be more fully shown, after it has been brought to a stand, be excited by a cause that acts primarily and directly upon the capillaries of these organs, and that can excite no influence upon the blood contained in the vessels of the brain or *medulla oblongata*, namely, artificial respiration. By which means also the circulation may easily be kept up for a considerable length of time through the lungs after all influence of the nervous centres has been entirely removed, as when the animal has been pithed. It is, moreover, opposed by the pathological fact, that a considerable degree of congestion may exist in the pulmonary vessels, before the blood is sufficiently carbonized to give rise to any diminution in the activity of the functions of the nervous centres.

As the doctrine under consideration is, as I understand, entertained by several physiologists in this country, to whose opinions much deference is due, and as it is of much importance in a practical as well as of interest in a scientific point of view, to ascertain its correctness, I determined, in order to do this, to put it to the test of experiment in such a way that arterial blood might be made to circulate through the brain and *medulla oblongata* of a dog after all access of air to the lungs had been prevented by the closure of his trachea. In which case the stagnation of the blood in the pulmonic capillaries ought not to occur if it were dependent upon the want of proper nervous influence being communicated to these vessels; the brain and nervous centres being properly stimulated by arterial blood.

Exp. 9. Three mongrel terriers, A, B, and C, were properly secured in such a way that their heads might be brought into close apposition. A tube, furnished with a stop-cock, was then introduced

into the proximal end of the left carotid artery of A, and another into that of the right carotid artery of C. These vessels had previously been ligtatured beyond the point at which the pipes were introduced so that no hæmorrhage might occur. The force of the heart's impulse, in the lateral dogs, A and C, was now measured by the hæmadynamometer, and found to amount to from $4\frac{1}{2}$ to 5 inches in each of them. A tube, furnished with a stop-cock, was next adapted to the trachea of the centre dog, B, and a pipe was introduced into the distal extremities of both its carotid arteries, which were tied below the point at which the pipes were inserted. The animal did not appear in the least to suffer from the ligature of both these arteries. One of the jugular veins of the centre dog was then exposed, and a ligature was passed under it in order that it might be punctured, so as to avoid the occurrence of plethora and apoplexy, when the carotid arteries of the two lateral dogs were connected with the corresponding vessels of the central one. The pipes in the carotid artery of A and C were then adapted by means of connecting pieces to those in the central dog, and were besides tightly tied together, so that they could not slip during the struggles of the animal. When this arrangement had been properly and securely made, the trachea of the centre dog was closed, the jugular vein was punctured, and the stop-cocks connecting its carotid arteries with those of the lateral dogs were opened. As soon as this had been done the vertebrals of the dog, B, (the centre one), were compressed with the fingers of an assistant, in order that the circulation through the brain might be confined as nearly as possible to arterial blood. The centre dog remained quiet for about a minute and a quarter; it then began to struggle, and in three minutes all movement had ceased, and animal life was extinct. The distal extremities of the carotid arteries of the centre dog were then examined, as they had been several times during the experiment, and were found still to pulsate, although somewhat feebly, from the impulse of the blood sent direct from the hearts of the lateral dogs. Nearly a pint of blood had flowed from the jugular vein during the experiment, so that the animal had clearly not died from plethora. The lateral dogs were both alive, but evidently enfeebled by loss of blood; and on the pressure in the carotid artery of one of them being measured by the hæmadynamometer, it was found to amount to not more than 3 or $3\frac{1}{4}$ inches of mercury.

The centre dog was opened about ten minutes after its death, by which time the action of the heart had entirely ceased. The lungs were of a gray mottled colour, and much congested, dark drops of blood exuding when they were cut into. The coronary vessels were quite black; the right auricle and ventricle and pulmonary artery were distended with black blood. The *venæ cavæ*

and liver were also much engorged. The pulmonary veins, and the left auricle and ventricle, also contained some black blood; the aorta was nearly empty, being merely moistened with a little blood.

In this experiment every possible precaution was taken to avoid those sources of fallacy that were likely to occur, and it was found that the animal died as quickly after the closure of the trachea, when arterial blood was circulating through the brain and *medulla oblongata*, as when these parts were, as in ordinary cases of asphyxia, permeated with venous blood.

The principal source of error in an experiment of this kind would necessarily arise from the supervention of apoplexy in the central dog, in consequence of its vessels becoming inordinately distended by the quantity of blood sent directly into them from the hearts of the two lateral ones, which accident actually occurred, (as Dr Kay has pointed out,) in a somewhat similar experiment performed by Bichat. The supervention of this accident was, however, guarded against in the present instance, by puncturing the jugular vein of the centre dog, before the stop-cocks were opened, and the blood began to flow into its carotids; and the utility of this precaution may be judged of by the fact, that nearly a pound of blood escaped by the puncture thus made, and which was doubtless almost entirely furnished by the lateral dogs. For when the pressure in the carotid artery of one of these was measured after the conclusion of the experiment, it was found that the column of mercury in the hæmadynamometer stood one and a-half inch lower than before the commencement of the experiment, showing clearly that the animal had suffered much from hæmorrhage.

That the centre dog did not die of hæmorrhage from the jugular vein was evident from the results of the *post mortem* examination.

It may be objected to this experiment that the venous blood conveyed by the vertebral arteries to the brain of the dog that was asphyxiated might have been sufficient of itself to suspend the nervous influence. But this could not have been the case; for during the experiment the vertebrals were compressed in such a way as to confine the cerebral circulation as much as possible to arterial blood. And even if this were not accomplished in a very perfect manner, yet the quantity of venous blood transmitted to the brain and *medulla oblongata* would not exceed that of the arterial transmitted by the carotids; and this mixed fluid would contain fully sufficient red blood to support the nervous influence in a diminished degree perhaps, but still to such an extent as to retard very considerably the progress of the asphyxia, which did not happen; the usual phenomena of that condition presenting themselves in the exact order and at about the average time at

which I have found them to occur in the other experiments that I have performed on this subject, and not being retarded in any very appreciable manner.

It is true that an opposite experiment to this has been performed by Bichat, the results of which might appear to be irreconcileable with those of the one just related; but a brief examination will, I think, convince us that this difference is rather apparent than real. I allude to the experiment in which that eminent physiologist found, that when the carotid arteries of two dogs were united by means of a canula, so that the heart of one might be made to propel blood into the brain of the other, if the dog that was propelling the blood were asphyxiated, the one through whose brain the venous blood circulated became confused, agitated, and lost the use of its external senses, and, if the experiment were continued, would die. More rapid and marked results were found to ensue when venous blood was injected into the carotid artery by means of a syringe. Dr Kay has, however, clearly and incontestably proved that the deleterious effects manifested in the first of these experiments are attributable to the receiving dog becoming plethoric; that the effects, which were slow in manifesting themselves, resemble those that follow too large an injection of blood into the veins of an animal; and that the very rapid death of the animal, when venous blood was thrown in by means of a syringe, was due to the injury that the texture of the brain received from the force employed.

I do not, however, wish it to be understood from the remarks that I have just made, that I deny the possibility of an animal being readily and speedily asphyxiated by the circulation of venous blood through the brain and *medulla oblongata.* But this would be in consequence of the suspension of the function of these parts breaking one of the links in that chain of vital actions, the integrity of which is necessary for the due maintenance of the respiratory movements, the imperfect performance of which would necessarily occasion an interruption to the chemical changes between the blood and the air in the lungs; on the cessation of which asphyxia must necessarily ensue. I think, then, from the experiment that has just been related, as well as from the arguments that have been adduced, we may conclude that the usual phenomena of asphyxia occur when the access of air to the lungs is prevented, whether the "nervous influence" be maintained or not.

β. We will now proceed to the examination of the opinion that is entertained by Dr Alison and others, namely, that the blood stagnates in the lungs in asphyxia, in consequence of the cessation of the vital attractions connected with the chemical changes that take place between the blood and the air in the lungs.

It will not be necessary for us to enter upon the general question of the dependence, in whole or in part, of the capillary circulation on the persistance of the vital attractions and repulsions between the blood and these vessels,—a doctrine which, as is will known, is advocated by several physiologists whose opinions are entitled to much weight, but merely to discuss this doctrine in its bearings on the subject of the present inquiry*.

Although there can be no doubt that the smaller vessels possess a distributive power over the blood, one by which they can modify the flow of blood to particular parts independently of the action of the heart, yet I do not think that we are warranted in supposing that any vital actions going on between the blood and these vessels can exercise a propulsive influence upon that fluid.

The following experiments, performed by Dr Sharpey, clearly prove that the force of the heart's contractions are of themselves fully sufficient for carrying on the circulation without the necessity of assuming the operation of minor causes.† A syringe having a hæmadynamometer attached was adapted to the thoracic portion of the aorta of a recently killed dog, which vessel had previously been ligatured immediately above the renal arteries, and the *vena cava inferior* was opened where it passes through the diaphragm. Beat bullock's blood was then injected and passed out of the *vena cava inferior*, after traversing the double capillary system of the intestines and liver, in a free stream with a pressure of $3\frac{1}{2}$ inches of mercury; when a pressure of 5 inches was used it flowed out in a full jet.

When the aorta was not ligatured above the renal arteries, but left pervious so that the blood might flow through the lower extremities, the same pressure was fully sufficient to drive the blood freely through their extensive vascular ramifications.

If the same instrument be adapted to the pulmonary artery, it will be found that a pressure of from $1\frac{1}{2}$ to 2 inches of mercury will suffice to drive beat bullock's blood through the capillaries of that organ, so that it may flow in a free stream from the left auricle or the pulmonary veins.‡

In the first two experiments just related, the force that sufficed to drive beat bullock's blood through the capillaries of the chylopoietic and portal systems was about the same as what is exercised by the left ventricle, certainly not more. In forcing the blood through the pulmonic capillaries much less power was required; proportionately probably to the strength of the impulse

* I would refer those who may wish to see this matter fully discussed, to Dr Allen Thomson's excellent Essay on the Circulation, in the Cyclopædia of Anatomy and Physiology.

† I may take this opportunity of stating, what I omitted to do on a former occasion, that these experiments were first suggested and performed by Dr Sharpey.

‡ In performing these experiments it is necessary that the bullock's blood be previously strained through a thick cloth, in order to remove any small coagula that may be present.

of the right ventricle. But as I have elsewhere remarked,* it would be exceedingly difficult, if not impossible, to prove the amount of propulsive power exerted by the right ventricle, as the experiment of attaching a hæmadynamometer to the pulmonary artery of a living animal would be attended by so many modifying circumstances, as not to admit of any conclusive result being obtained. If, however, we are allowed to assume that a muscle contracts with a power proportioned to its size, we shall, as the thickness of the walls of the right ventricle are to those of the left, according to Bouillaud, as $2\frac{1}{2}$ to 7, be allowed to estimate the power of its contractions in the same ratio. Now, as the left ventricle propels the blood in the arterial system with a pressure equal, on an average, to about five inches of mercury, if any thing, rather less, certainly not more, the ratio of the pressure exercised by the left ventricle as compared to that of the right, may be stated by the formula $7 : 2.5 :: 5 : 1.78$; which would probably be about the pressure in the pulmonary artery, and which agrees with the force required to propel bullock's blood through the vessels of the lungs, which amounts, as has already been stated, to from $1\frac{1}{2}$ to 2 inches of mercury. Although these experiments are not conclusive evidence against the existence of any motor power in the circulation besides the heart, yet as they show that the pressure exercised by that organ would of itself be fully sufficient for the purpose of carrying on the circulation, they may be considered as affording strong presumptive evidence in favour of the opinion, that the action of the heart, aided by the distributive power of the arteries, is of itself sufficient for carrying on the circulation of the blood.

But instead of arguing upon the general question of the dependence of the capillary circulation on the persistance of the vital actions connected with the chemical changes, let us inquire, whether, even were this point conceded, it would be sufficient to account for the obstruction to the passage of the blood through the capillaries in asphyxia. If the arrest of the circulation in these vessels were owing to the cessation of the chemical changes, it must necessarily follow that, when these cease, the passage of the blood must be arrested. Now this is not the case in asphyxia. The chemical changes between the blood and the air in the lungs cease, and the blood in the arterial system becomes perfectly venous in less than two minutes after the access of air has been cut off. Bichat states that the blood in the arteries assumes a perfectly venous character in from a minute and a half to two minutes, in which statement Dr J. Reid coincides. I have frequently noticed the blood in an exposed artery to be perfectly black and quite undistinguishable from venous blood in a minute and a half; and occasionally even

* Edin. Med. and Surgical Journal, January 1844.

in a somewhat less time than this; the difference being probably owing to the quantity of residual air that happens to be in the lungs when the trachea is closed. But at all events, most writers on this disease agree, that in less than two minutes the blood is perfectly black, and we may accordingly take this as the period at which the chemical changes have ceased. The circulation of this black blood, however, continues for a very much longer time; and not only continues, but goes on actively and forcibly. I have found the average time at which the pulsation in the femoral artery ceases to be perceptible, to be the eighth minute after the cessation of respiration; that is to say, at least six minutes after the blood has become perfectly venous; and it is highly probable that, although the pulsations may no longer be perceptible in an artery so far removed from the centre of the circulation as the femoral, that the blood still continues to circulate through the lungs for some time after this. Indeed this is rendered highly probable by an experiment that I shall immediately relate. The evidence of Dr Alison* on this point is very distinct. He says, "the circulation through the heart has very generally ceased within less than ten minutes from the commencement of the obstruction." In the table of one of Dr Reid's experiments on the pressure in the arterial system, it is stated, that at the sixth minute after the trachea was shut, the mercury oscillated between 3 and 6 inches, thus showing that the circulation was going on actively. From this, then, it is very evident that the circulation continues for at least from six to seven minutes, and must take place seven or eight times after the cessation of the chemical changes; which fact appears to me to be inconsistent with the idea that the passage of the blood through the capillaries is dependent upon the vital actions connected with these changes, more particularly when we recollect that the force of the heart's action is at the same time diminished.

The following experiment will show that, provided the heart's action be maintained, the circulation of black blood may continue through the lung for a considerable time beyond the period at which the circulation ordinarily ceases in asphyxia, and that the cessation of the circulation must consequently depend in a great measure on the failure of that action.

Exp. 10. A tube was adapted to the trachea of a young spaniel. The animal was immediately pithed; artificial respiration was then set up so as to maintain the heart's action, and the chest was laid open. A ligature was next passed under the right bronchus close by the bifurcation of the trachea, and tied tightly so as to prevent the entrance of any air into the right lung. This was done in about three minutes from the time that the animal was pithed. The in-

* Art. Asphyxia, Cyclop. Anatomy and Physiology.

flation, which was only carried on by the left lung, was now continued,—the right lung remaining in a mid state between collapse and distension, and quite motionless. The heart was beating tumultuously about 120 per minute.

3d minute after bronchus was tied.—Heart beating from 100 to 120; still very irregularly and tumultuously.

9th minute after ligature of bronchus.—Heart beating forcibly, but rather irregularly, from 60 to 70. A ligature was now passed under one of the pulmonary veins of the right lung, (the obstructed one,) and tied as near as possible to its entrance into the auricle. The vessel was then punctured on the distal side of the ligature, and a jet of semi-venous blood escaped. The blood was not quite black, owing to regurgitation having taken place from the left auricle, as the pulmonary vein was not ligatured until after inflation had been established for several minutes.

11th minute.—Heart beating 45 to 50 more regularly. Inflation of left lung continued. A quantity of perfectly venous blood trickles out of the puncture in the pulmonary vein; a fresh portion collecting and trickling slowly down whenever the puncture was wiped.

14th minute.—Heart beating 45. The black blood continues to ooze from the puncture in the pulmonary vein.

17th minute.—Much the same as at last report, the blood continuing to ooze slowly but very distinctly. During the whole of this time not a bubble of air gained admission into the right lung, and the blood that oozed out of the puncture was perfectly black. Experiment discontinued.

On examination, the pulmonary veins of the left lung and the left auricle were found filled with bright arterial blood, a small quantity of which was also contained in the left ventricle.

Exp. 11. In another experiment of the same kind.—At the tenth minute after the bronchus had been tied, the heart was beating 60 to 64 strongly and regularly; artificial respiration was kept up with the left lung alone, the right one being quite collapsed.

14th minute.—A ligature having been passed under a pulmonary vein of each lung, these vessels were punctured on their distal side. A quantity of black blood flowed from the pulmonary vein of the right lung, whilst from the corresponding vessel of the left lung florid arterial blood escaped in a small jet.

17th minute.—Heart beating about 20 per minute. From the pulmonary vein of the right side dark blood is still flowing, rather slowly, but very distinctly; whilst from that of the left lung a larger quantity of arterial blood is evidently escaping.

19th minute.—Heart 14 to 16; flow of blood still continues, but very slowly.

21th minute.—Ventricular action and the flow of blood from the pulmonary veins have ceased.

The blood that had accumulated in either side of the chest was now carefully collected and measured, when it was found that that which had flowed from the right lung amounted to $2\frac{1}{4}$ drachms, whilst that from the left amounted to $3\frac{1}{2}$ drachms; and in another experiment of the same kind the quantities that escaped were respectively $2\frac{3}{4}$ and 4 drachms.*

From these experiments it is evident that, provided the heart's action be maintained, black blood may be made to circulate through a lung (in which the chemical changes have entirely ceased), for a much longer period than in ordinary cases of asphyxia; the same force that keeps up the circulation of red blood in one lung sufficing for that of black blood in the other; and this although the force of the heart's contractions must be somewhat enfeebled in consequence of the deficient aëration of the blood when one lung is useless. It is true that the quantity of blood that is sent through the two lungs is not the same, more passing through the left in which respiration is carried on than through the right in which it is arrested; the difference between the two quantities depending probably on the arrest of the respiratory movements, and on the obstructive cause in the pulmonary vessels in the right lung. But yet a considerable quantity passes through, and continues to do so until the impulse of the heart has become so much enfeebled as no longer to have the power to propel the blood through so extensive a vascular system as that of the lungs, and to overcome the obstruction that exists in the vessels of that organ.

That the heart's action is, of itself, sufficient, when vigorous, to keep up the circulation through a lung in which the chemical changes have ceased, and through which dark blood must consequently be passing, is evident from the fact of the circulation continuing actively in lungs that are compressed by effusion into the pleural sac, whether natural, or, as Goodwyn has observed, when artificially induced. In hydrothorax there is not any difficulty in the transmission of blood through the compressed lung, although in this case, as in the experiments just related, the heart's action can hardly be supposed to be as vigorous as usual, inasmuch as the oxygenation of the blood can be but imperfectly performed, one lung only being in action.

The influence of artificial respiration in re-exciting the circulation through the lungs, after the action of the heart has ceased, is generally advanced as an additional argument in favour of the dependence of the circulation through the pulmo-

* This experiment is rather difficult of performance, as the bronchus of the dog is very short, and there is always the danger of the heart's action becoming very much enfeebled, if not entirely arrested before that tube can be ligatured. It is therefore advisable to use young dogs, as the irritability of the heart prevails for a longer time in them than in the older ones.

nary capillaries on the persistance of the vital actions connected with the chemical changes. But if this fact be more closely examined, it will be found that it affords no evidence in favour of this doctrine. It has already been stated as one of the proofs of the existence of an obstruction to the passage of the blood through the capillaries of the lungs, that the pulmonary artery is, in cases of death by asphyxia, invariably found to be in a state of tension from the accumulation of blood in it. This tension is very considerable, so much so, that if, after death, when the heart's action has entirely ceased, a small puncture be made in the vessel, the blood will spout out with some degree of force. When artificial respiration, therefore, is set up, and the venous blood that has accumulated in the lungs has became properly aërated, the tensive condition of the trunk and branches of the pulmonary artery will act as a *vis-a-tergo*, and will propel the newly oxygenized blood into the pulmonary veins and left auricle; the pulmonary artery being always found, after artificial respiration has been continued for a few minutes, flaccid, and emptied of much of its blood. In whatever way, then, the restoration of the chemical changes act in preparing the blood or the smaller vessels for the re-establishment of the pulmonic circulation, and in removing the obstruction to the passage of the blood, an impulse is communicated to that fluid by the *vis-a-tergo* influence of the tension of the pulmonary artery, which will be fully sufficient to propel it into the left side of the heart, and thus (as will hereafter more fully be shown) to re-excite the contractions of that organ.

For the reasons, then, that have just been stated, as well as on account of the uncertainty of the general doctrine of the dependence of the capillary circulation on vital attractions and repulsions between the blood and the vessels connected with the chemical changes, we cannot coincide with the opinion that would refer the stagnation of the blood in the lungs in asphyxia to the cessation of these actions, and must accordingly seek another mode of explaining this remarkable phenomenon.

γ. The next opinion is the one that is advocated by Dr Kay, who supposes that, in consequence of the non-arterialization of the blood, the minute pulmonary vessels, which usually convey arterial, become incapable of transmitting venous blood, which consequently stagnates in the lungs.

If by the "smaller pulmonary vessels," Dr Kay means the capillaries, his explanation cannot, for reasons that will by and by be stated, hold good.

But although the obstruction cannot be supposed to exist in the capillary vessels, may it not reside in the smaller ramifications of the arterial system, including of course the pulmonary veins, as

conveying red blood? This opinion, which has, I believe, been for some time entertained, though not, as far as I know, published by my friend Dr C. J. B. Williams, a gentleman as distinguished for his physiological as for his practical knowledge, appears to me to be the correct one.

The experiments of Dr J. Thomson, Dr Wilson-Philip, Dr Hastings, Dr C. J. B. Williams, Kaltenbrunner, and Wedemeyer, have indubitably proved that the arteries possess a contractile power; and the observations of Hales and of Wedemeyer farther tend to show that this contractility may be excited by the injection through them of fluids of a more or less stimulating character. The supposition is therefore a natural one, that the venous blood which circulates in asphyxia, may act as a stimulus to the smaller divisions of the arterial system, exciting the contractility of these vessels, and that thus the obstruction in the circulation may be occasioned. This, I think, will on closer inquiry prove to be really the fact.

In this way we should be able to explain the continuance of the circulation for a considerable period after the chemical changes have ceased, and, indeed, as long as the heart has power to maintain it. For the contractility that takes place in arteries on the application of a stimulus is a slow process. Dr J. Thomson observes, that though arteries are contractile to a stimulus, a period of from one to three minutes elapses before the contraction begins; and hence we can explain how it is that the obstruction only comes on gradually, and that the circulation is enabled to continue, which, as has already been stated, ought not to be the case if its arrest depended on the cessation of the vital actions connected with the chemical changes.

It may be as well, before proceeding any farther, to notice here the arguments that have been brought against the doctrine under consideration. With one exception these apply to the capillaries and not to the smaller divisions of the arterial system. It is very truly stated by Dr Alison, that the stagnation cannot arise from the stimulus of venous blood being insufficient to excite the capillaries, as it has not been proved that these vessels are irritable, nor that they possess coats. This, however, does not hold good with regard to the smaller arterial ramifications, which, as has already been stated, have been fully proved to possess a contractile power. But, it is argued by Drs Alison and Carpenter, if the stagnation of the blood in the smaller divisions of the pulmonary artery, or in the capillaries of the lungs, depended on any vital action possessed by these vessels, it would follow that venous blood, which makes its way through them so slowly, must be a powerful stimulus to them, and that arterial blood, which is so readily transmitted, must act as a sedative; which is opposed to the fact that

arterial blood and the oxygenized fluids generally possess in every other known instance the stimulating, and venous blood and the carbonized fluids the sedative power.

This argument, however plausible it may be, is very far from being conclusive. In the first place, the same substance may act as a sedative to one set of organs or tissues and as a stimulus to another. Dr Williams* very justly observes, "the elementary action of ice-cold water on the arteries is strictly stimulant; exciting their vital property of contraction; but its operation on textures and organs is sedative, because it impairs the circulation which supports their functions." And again, "Some substances which act as stimulants to the heart and vessels, and to the cerebral functions, operate as sedatives to the medullary system; these are the stimulant antispasmodics . . . . . they probably operate by giving vigour and equality to the circulation." These observations bear directly on the point in question, and prove clearly that the expressions "stimulant" and "sedative" are merely relative and not positive, and that the cause of the sedative action of one agent on one set of tissues or organs may merely be the effect of its primarily having exercised a stimulant influence on another. But we have distinct evidence of carbonic acid occasionally acting as a stimulant; for when an attempt is made to inspire pure carbonic acid violent spasm of the glottis takes place, which prevents the gas from entering the lungs.† In this case it is clear that carbonic acid acts as the stimulant, and the oxygenized air, so to speak, as the sedative agent.

We find, besides, that every part of the body is provided with a special sensibility, "which determines," to use the words of Bichat, "the nature of its relations with those foreign bodies that may happen to be in contact with it." It is in this way that the peculiar sensibility of the pancreatic, salivary, and parotid ducts, of the *ductus communis choledochus*, and, in a word, of those of all the excretory organs generally, is in exact relation with the nature of their own secretions, but not with heterologous fluids; so that it does not allow these to enter them; and when in their passage before them any particles of a foreign fluid happen to find their way into the ducts they immediately give rise to spasm and contraction. It is for this reason that the excretory ducts, although opening on the mucous surfaces, and in contact with a number of different secretions and substances, never admit any of them,—that the chyme can never find its way into the *ductus communis choledochus*, although its particles would readily be admitted by the mouth of that duct. It is in the same way that we must explain the fact that the absorbent action of the lymphatics is confined to

* Principles of Medicine, pp. 33 and 72.

† Turner's Elements of Chemistry. 4th Ed. p. 267.

particular fluids, some foreign matters being taken up by them although wth difficulty, whilst others are not absorbed at all;—that the mucous membrane of the eye is irritated by a drop of urine or of bile, which have no influence on that of the kidneys or gall-bladder, and that the mucous membrane of the stomach is not sensible to the presence of a foreign body, whilst that of the bladder would be violently irritated by it. These examples might be multiplied to an almost indefinite extent, but a sufficient number have been adduced to show how universally a special sensibility excited by the presence of a heterologous substance is possessed by the tissues of the animal body, and that the agent that calls this sensibility into action need not be a stimulant in the ordinary sense of the word, for it to possess an excitant influence upon a tissue that is unaccustomed to its presence.

That the same property is possessed by the arterial system, has been proved by Hales and Wedemeyer, who, as it has already been stated, have found that stimulating liquids were obstructed in their passage through these vessels. The question, therefore, arises, whether venous blood ought to be considered a stimulant to this system, including, of course, the pulmonary veins. It has already been shown that the term stimulant is a relative one, that it is usually sufficient for a fluid to be a heterologous one for it to possess stimulating properties on the tissue or part to which it is applied, and that merely because it acts as a sedative on one organ, it does not follow that it may not have a stimulant action on another. On reasoning from analogy, therefore, we should be disposed to look upon venous blood as a stimulant to the arterial system, that is, as having a tendency to excite the contractility of these vessels.

But we may go a step farther, and prove that it actually possesses this power; causing these vessels to contract distinctly, as I have several times observed, on examining, under the microscope, the mesentery of rabbits during and immediately after the process of asphyxia. This may be done without much difficulty, as the circulation in these animals, when quite young, continues for many minutes after the struggles of asphyxia have ceased. On asphyxiating a young rabbit, a portion of whose mesentery has been conveniently fixed under a powerful microscope, the following phenomena will be observed to ensue. For about a minute after the struggles of the animal have ceased the circulation appears to be going on with its usual rapidity; it then gradually becomes somewhat slower, the arteries contracting in size, containing less blood, and assuming a lighter and more tawny colour than before, whilst the veins become congested and evidently fuller, assuming, when viewed by transmitted light, a very beautiful crimson hue. As the circulation becomes more languid the arteries continue con-

tracting, and acquire a lighter colour, the diminution in their size, and the difference in the quantity of blood contained in them and in the veins being most marked. The motion of the blood in the capillaries now becomes oscillatory, the whole mass of the blood being at each impulse of the heart slowly propelled forwards, and then moving backwards. This to and fro motion continues for some little time, and then ceases entirely. When the circulation has been completely brought to a stand, the arteries may be observed to be much contracted and flattened, of a light tawny colour, and offering the most marked contrast to the appearance of the veins, which are evidently much distended, and as I have just said, when viewed by transmitted light, of a most beautiful crimson colour. On restoring the heart's action by setting up artificial respiration, an impulse was evidently transmitted from the blood in the arteries to that in the capillaries, in a pulsatory and jerking manner, which was soon communicated to the veins, driving forward the whole mass of globules accumulated in them ; and gradually becoming more equable and powerful, until the circulation was completely restored. I have watched these phenomena most attentively in the mesenteries of young rabbits, and have never observed any thing like spontaneous movements in the capillaries ; the blood contained in which was invariably most clearly and distinctly influenced solely by the impulses it received from that which was contained in the arteries. Nor have I ever been able to discover any obstruction in the vessels, in consequence of the adhesion of colourless globules to their sides,—a phenomenon that I especially watched for, and which has, by several, been supposed to occur. The diminution in the diameter of the smaller arteries, and the proportionate difference between them and the neighbouring veins was most evident, and was such as could leave no doubt on my mind as to the important part that the contractions of these vessels plays in giving rise to an obstruction to the passage of the blood through them in asphyxia ; in which I have no doubt that it is the principal, if not the sole agent.

I do not feel myself called upon to make any remarks on the cause of the arrestment of the sensorial functions in asphyxia, as that subject has already been fully and ably investigated by Dr Reid. Bichat's opinion, that the immediate cause of the suspension of these functions was due to the noxious influence of the venous blood circulating through the cerebral vessels, was disputed by Dr Kay, who endeavoured to prove that this condition is rather attributable to the diminution in the quantity than to any alteration in the quality of the blood circulating through the brain. Dr Reid showed the fallacy of the experiments that were adduced by Dr Kay in support of his doctrine, and proved satisfactorily that the suspension of the functions of the encephalon is chiefly,

if not entirely, dependent upon the circulation of venous blood through its arteries. Not that the venous blood exercises any positively deleterious influence, but rather that it acts by the deprivation of the natural stimulus of arterial blood; the functions of the brain rapidly manifesting themselves when arterial blood is readmitted, provided this be done in a given time. It has likewise been proved by this physiologist that the functions of the cerebral hemispheres are annihilated a short time before those of the *medulla oblongata*, the respiratory movements continuing for a short time after the animal has become quite insensible. I may state generally, without analyzing the evidence on which these statements are founded, that it is in complete accordance with what I have had frequent occasion to observe in experiments on asphyxia.

The mode in which death occurs, and the order in which the vital functions cease in asphyxia, appear to be as follow:— The blood, whilst it is assuming a venous character, passes for some time with tolerable freedom through the vessels of the lungs into the left side of the heart, whence it is propelled through the whole system, causing first a derangement and then a suspension of the functions of the sensorium, so that the animal becomes more and more insensible; perfect unconsciousness and cessation of all voluntary movement occurring in about a minute and a half or two minutes. The functions of the *medulla oblongata* are continued for some little time longer. The blood having now become perfectly venous begins to be obstructed in its passage through the ultimate ramifications of the pulmonary veins and of the arterial system generally, occasioning congestions of the pulmonary artery, of the right cavities of the heart, and of the whole of the venous system, and the quantity sent to the left side of the heart becomes materially lessened. The obstruction in the vessels goes on increasing, and coincident with this is a diminution in the activity of the heart's action, which becomes both slower and feebler in consequence of the lessened quantity and altered quality of the blood that finds its way into the left ventricle and that is propelled into the coronary arteries, until at last, from a concurrence of these several causes, stagnation of the blood in the vessels of the lungs, diminution in the quantity, and alteration in the quality of the blood supplied to the substance of the heart, and distension of the right auricle and ventricle, this organ ceases to contract, and organic life becomes extinct.

Before concluding this part of the paper, it may be as well to mention the average period at which I have found, in the course of a considerable number of observations, some of the principal phenomena of asphyxia occur.

Voluntary movements cease in . . $1\frac{3}{4}$ minutes.

Involuntary movements in . . $2\frac{1}{2}$ minutes.
Blood in arteries becomes as black as that in the veins in . . . $1\frac{3}{4}$
Occasionally as early as . . . $1\frac{1}{4}$
Contractions of the ventricles cease in . $9\frac{1}{2}$
The earliest that I have observed has been in $6\frac{1}{2}$
The latest in adult animals in . . 14
Twitchings and irregular movements continue for some little time longer.
The left auricle on an average in . 12
I have seen it in an adult animal continue to contract till the . . . 37th
The right auricle on an average in $19\frac{1}{2}$
I have seen it in an adult animal continue to contract till the . . 44th
Pulsations continue in the femoral artery on an average for . . . $\frac{1}{2}$

In very young animals the time that the contractility of different parts of the muscular system is maintained is very different.
In puppies four days old movements continued for 16 minutes.
Ventricles continued acting regularly for . 1 hour 57
But twitchings and irregular movements continued for . . . 3 hours 4
Auricles continued acting for . 3 hours 25

The points, then, that I have more particularly endeavoured to establish in this part of the paper are,

1*st*, That although the persistance of the respiratory movements has some influence in maintaining the circulation through the lungs, yet that their arrest is not by any means the sole cause of the cessation of the circulation.

2*d*, That a diminution in the force and frequency of the contractions of the heart consequent upon the altered quality and lessened quantity of the blood circulating through its muscular substance, is one of the principal causes of the cessation of the circulation in asphyxia, as is evident from the fact that, when the force of the heart's contractions is maintained by a supply of arterial blood to its muscular substance, it is enabled to propel black blood through a collapsed lung.

3*d*, That the obstruction which has been proved to take place in the pulmonary and systemic circulation is due to the venous blood exciting the contractility of the minute divisions of the arteries and pulmonary veins by acting upon their special sensibility.

4*th*, That the cause of the stoppage of the circulation in asphyxia is therefore threefold; depending, 1. Upon the arrest of the respiratory movements; 2. Upon the weakening of the heart's action; and 3. Upon the obstruction offered to the blood (pro-

pelled with diminished force) by the refusal of the pulmonary veins and minute arteries to receive venous blood.

Treatment.—We will now proceed to the consideration of some of the more important points connected with the treatment of asphyxia; and in so doing it is not my intention to give a history of the subject, or even a resumé of the principal plans that have been recommended for the recovery of persons labouring under the disease in question, but rather to confine the remarks that I have to make to the discussion of some of the points that are more particularly insisted upon by the Royal Humane Society, and to suggest some new or modified plans of treatment that will, I hope, be found to prove serviceable in particular cases of asphyxia.

Before entering more at length upon this subject, it will be better to understand clearly the principal objects that ought to be fulfilled in the treatment of asphyxia. It has been shown in the preceding pages that one of the chief causes of the arrest of the circulation in this disease is the obstacle offered to the passage of the blood through the lungs consequent upon the cessation of its arterialization and the arrest of the respiratory movements, and that the contractions of the heart cease, and the functions of the brain and nervous centres become suspended, in consequence of the circulation of unoxygenized blood, deficient in stimulating and nutritious properties, through their tissues. Our first object should therefore be to arterialize the blood, and our second to restore or maintain the action of the heart, and thus cause the freshly aërated blood to circulate through the nervous centres, on which it acts as the most powerful stimulant, re-exciting the functions of the *medulla oblongata* and the nerves of respiration, and thus enabling the respiratory movements to be properly maintained, and consequently the oxygenation of the blood to be naturally performed.

Such being the principal indications to be accomplished in the treatment of asphyxia generally, we will now proceed to the consideration of the most efficient mode of carrying them out in those cases of suspended animation that proceed from drowning, which, as being the most frequent, are the most important to be properly understood.

As it is not necessary in every stage of asphyxia to have recourse to the same plan of treatment, I think that it will tend to place this subject in a clearer light if we divide the cases of this disease into the two following classes:—

1*st*, Those in which the action of the heart is still continuing, however feebly.

2*d*, Those in which the action of the heart has ceased.

It would be a matter of some importance in the treatment of asphyxia if the average time at which the heart ceases to beat could be ascertained with any degree of accuracy. This it is, however, excedingly difficult, if not impossible to do, as the time may be much prolonged by the existence of a number of modifying circumstances, such as the quantity of air that may be contained in the lungs at the time of submersion—whether the sufferer struggles much, or remains quiet, (muscular movements having a tendency to cause more rapid consumption of the oxygen), his age and strength, and lastly, the condition of his nervous system. For if fainting ensue, in consequence of the mental shock experienced at the moment of immersion, submersion may last a very considerable time, and the actions of the heart will still continue,—the person suffering from syncope rather than from asphyxia. The discrepancies that occur in the statements of authors, as to the length of time that recovery is possible after submersion, are no doubt attributable to these two conditions—syncope and asphyxia—having been confounded together; insensibility, which is common to both, being the chief symptom that has been taken into account.

As syncope appears to be a common consequence of submersion, it is necessarily a point of some importance to inquire into the physiological differences between that condition and asphyxia. In syncope the primary impression is upon the nervous system, and the heart and circulatory apparatus are secondarily affected. The motions of the heart become much enfeebled, but still they continue; and a certain quantity of blood, small indeed in amount, continues to circulate through the lungs, consuming slowly the oxygen of the residual air left in them, which appears to be sufficient for the maintenance, for a considerable time, of the diminished vitality of the whole system. In this respect a person in a prolonged syncope resembles an animal during hybernation; the functional activity of the cerebrum appearing to be suspended, and animal life to be extinct; but yet the organic existence is prolonged, though in a feeble and imperfect manner. In asphyxia, on the other hand, the primary disorder is to be looked for in the respiratory and circulating systems, the brain and *medulla oblongata* being only secondarily affected. The blood rapidly assuming a venous character, becomes incapable of affording a proper stimulus to the heart, and being arrested, partly from the want of a due propulsive power in that organ, and partly from its inability to pass through the smaller arterial ramifications, the functions of organic life become extinct in the way that has already been explained. The pathological conditions of the system differing so materially in these two diseases, the treatment ought not, of course, to be directed to the same ends. In the one case, we should endeavour to reoxygenize the blood; in the other, to rouse the sensibility of the brain and *medulla oblongata*.

The difference in the appearance of a person in a state of syncope, and of one that is labouring under asphyxia, is equally well marked. In syncope the face is pale and bloodless, the features sunk and contracted, and the eyes partially or entirely closed. In asphyxia, on the contrary, the face is livid and bloated, the lips swollen, and the eyes open. But distinct as these appearances may be in pure and uncomplicated cases of each affection, yet they do frequently, in actual practice, run into one another, and are conjoined in greater or less proportion, so that they lose much of their value, and we are obliged to be guided in our treatment, rather by general rules than by an endeavour to ascertain how much of each case is referrible to asphyxia, and how much to syncope.

The essential practical difference between syncope and asphyxia is, that in the former case the contractions of the heart, though much enfeebled, may continue for a considerable, almost an indefinite, period; whereas in the latter they quickly cease. The precise time in which the heart ceases to beat in cases of unmixed asphyxia is very difficult to ascertain in man. In dogs, as has already been stated, the ventricular action continues on an average for $9\frac{1}{2}$, and occasionally for 14 minutes, the difference depending probably on the age of the animal, and I do not remember ever to have failed in re-exciting its action by the artificial inflation of the lungs at any period before the ventricular contractions had ceased; as I have frequently ascertained by opening the chest after the animal has been asphyxiated, but before the heart has ceased beating, and then inflating the lungs. In asphyxia from drowning, in man, unattended by syncope, I should suppose that the contractions of the ventricles have most generally ceased before five minutes have elapsed after complete submersion. My reason for this assumption, which it is exceedingly difficult to prove, is, that Mr Woolley, the surgeon to the Humane Society's Receiving-House in Hyde Park, and a gentleman of great experience in these matters, states, in the reports of that institution, that no cases were saved that had been more than four minutes under water. And I understand, on good authority, that it is very seldom that cases that have been less than four minutes under water are lost. Now, as the measures adopted by this Society do not act directly upon the blood-vessels of the lungs, or upon the heart, they can most probably only be successful whilst that organ is still pulsating, and will fail in all those cases in which the contractions of the heart have either entirely ceased, or have become so excessively enfeebled as to require the application of a direct stimulus, in order to re-excite them. It is true that, since this statement was made, two cases have been related in the reports of the Humane Society, one in that for the year 1840, another in that for 1843, in both of which the time of submersion appeared to amount to

five minutes. There were several circumstances attending these cases, however, that render them less important than they otherwise might have been in determining the point in question. Mr Woolley speaks thus of the first case: "Walsh was five minutes under water, and during the whole of that time the process of expelling air from the lungs was seen to go on. The lungs must, by the end of that time, have been in a state of collapse, and, on being taken out, the atmospheric pressure would necessarily fill with air the vacuum which had been formed, and the spontaneous return of respiration proved that there was nothing present in the lungs to prevent it." I shall by and by have occasion to advert again to this very interesting case; in the meantime I will content myself with pointing to the fact, that the process of respiration was spontaneously re-established; and that consequently this case can scarcely be adduced in illustration of the time that the organic functions cease in asphyxia, as the inspiratory efforts that were spontaneously made, might possibly, by arterializing a certain quantity of blood, have re-excited the contractions of the heart.

The second case, which occurred in June 1842, although most interesting in itself, is equally unsatisfactory as regards the point at issue; for, in relating it, the superintendent says, "Whilst cutting off her clothes, I heard for the first time a rattling in the chest, which I thought was an effort to breathe." In this instance, therefore, it is also possible that an artificial inspiration, spontaneously performed, re-excited or kept up the contractions of the heart.

As these cases cannot, therefore, as far as the point under discussion is concerned, justly be considered unequivocal exceptions to the fact deducible from Mr Woolley's remarks, I think that we are justified in taking four minutes as the minimum time that the heart continues to contract in cases of submersion; patients during that period being recoverable by means that do not directly influence it; such as the warm bath and friction.* When the contractions of that organ persist long after this, there is probably a state of syncope rather than of asphyxia; the sufferer having fainted at the moment of immersion, or very shortly afterwards, before there had been time for the process of asphyxia to make much progress.

We will now proceed to the consideration of the general plan of treatment that is to be adopted in those cases in which the period of submersion does not exceed four minutes. And here

* I have been informed by Mr Woolley that he believes that several other cases of recovery have occurred, in which the patient has been more than four minutes (in one case probably six minutes) under water; the men employed by the R.H.S. having been found to be in the habit of understating the time that the sufferers have been submersed. This, however, does not influence the statement about the minimum time at which the heart ceases to act.

it may be stated, that these constitute by far the largest majority of cases of drowning. Thus, for instance, in the Report of the Humane Society for 1840, it is stated that 185 cases were brought under the notice of that institution during the preceding year; and that, out of these, only sixteen were not recovered. Now, as it is stated in that very report, by the very intelligent and humane surgeon of the Receiving House in Hyde Park, that, with one exception, no case had been thus saved that had been more than four minutes under water, it must necessarily follow that at least 168 out of the 185 cases must have been submersed for a less period than four minutes.

The means recommended by the Humane Society for the recovery of persons from drowning, and employed at their institution in Hyde Park, appear to be well adapted for the treatment of this stage of asphyxia. They consist, after the nose and mouth have been cleared of any mucosities, in the application of heat by means of a bath at about the temperature of 100°, until the natural warmth is restored, in the employment of brisk friction, and in passing ammonia to and fro under the nostrils. It is evident that these measures can have no direct influence upon the heart and lungs; but can only act as general stimuli to the system, equalizing the circulation if it be still going on; and, by determining to the surface, tending to remove those congestions that are not so much the consequences of the asphyxia, as of the sojourn of the body for several minutes in cold water. They would, therefore, be of especial service during the colder seasons of the year. A hot bath may also, by the shock it gives, excite the reflex respiratory movements. With the view of doing this with a greater degree of certainty, I would suggest that cold water be sprinkled or dashed upon the face at the time that the body is immersed in the hot-bath, as in this way a most powerful exciting influence can be communicated to the respiratory muscles; and the first object of treatment in all cases of asphyxia, the re-establishment of respiration, would more rapidly and effectually be accomplished; deep gaspings ensuing, by which the air would be sucked into the remotest ramifications of the air-cells, arterializing the blood that had accumulated in the pulmonary vessels, enabling it to find its way to the left cavities of the heart, and thus to excite that organ to increased activity. The recommendations of the Humane Society appear to be most judicious, as far as regards those cases of asphyxia in which the sufferer has been but a short time submersed, and in which the heart is still acting, and the respiratory movements have either begun of their own accord on the patient being removed from the water, or in which they are capable of being excited by the shock of a hot-bath, aided, as I would suggest, by the dashing of cold water in the face. The

lungs should, at the same time, be inflated by compressing the chest and abdomen, so as to expel the vitiated air, and then allowing them to recover their usual dimensions by the natural resiliency of their parietes. A small quantity of air will, in this way, be sucked in, each time the chest is allowed to expand, and thus the re-establishment of the natural process of respiration may be much hastened. This simple mode of restoring the vital actions should never be omitted, as it is not attended with the least danger, and does not in any way interfere with the other measures employed. If, by these means, we succeed in restoring the proper action of the respiratory movements, it will merely be necessary to pay attention to some points of the after treatment that will presently be adverted to. Should we, however, fail in restoring respiration, we should have recourse to other means that will immediately be mentioned.

We now come to the treatment of the second class of cases, those in which the action of the heart has entirely ceased, or in which the sufferer has been submersed for more than four minutes.

When a person is withdrawn from the water, after having been immersed for a few minutes, the surface of the body is invariably cold and the vessels constricted. The first indication of treatment, therefore, that naturally presents itself is the application of warmth, so as to restore the natural temperature, and as soon as the circulation may be re-established, by determining to the surface, to relieve internal congestions. Two questions, however, present themselves *in limine.*

1*st*, What temperature is it advisable to employ?

2*d*, How is the warmth to be applied?

The intimate connection between animal heat and respiration is well known to all physiologists; the temperature of an animal being in proportion to the quantity of air that it consumes; and, (as every one knows,) in hybernation, a condition that presents many analogies with asphyxia, the respiratory function is much enfeebled, and the evolution of animal heat greatly diminished. These facts are so universally known, that it is needless for me to do more than allude to them as illustrations of the subject under consideration. Edwards has further shown that an increase of temperature compels an animal to consume more oxygen; that when the temperature of the medium in which an animal is placed is raised, the activity of its respiratory functions must be proportionally increased, and experiment has proved, that if the temperature of animals in a torpid condition be suddenly raised fatal results are very apt to follow. These facts are of much value in determining the degree of heat that ought to be applied to man when his vital actions are depressed. The Humane Society recommend a bath of a temperature of from 98° to 100°, or "as

hot as the hand can bear without pain," as well as hot bricks, bottles of hot water, or blankets wrung out of hot water. Now this degree of heat may perhaps be applied with safety to persons who have been immersed during the summer months, when the temperature of the water is between 50° and 60°; but if any dependence can be placed on the observations of Edwards, which are most trustworthy, it would unquestionably be far too high to be safely applied to persons who have been immersed during the winter, when the temperature of the water is perhaps but little above 32°. In this case I should certainly (being guided by those observations) recommend that the patient be not at first exposed to a greater degree of heat than from 85 to 90 degrees.

The next subject for consideration is, to say the least, an equally important one; I allude to the nature of the medium through which warmth is to be applied; and although the warm bath would appear to be the most efficacious means of accomplishing this, yet I perfectly agree with the opinion of Drs Kay and Carpenter that its employment is open to very serious objections, and is most decidedly opposed to two well-ascertained facts in physiology; namely, that the integument, even in man, acts as a respiratory organ, and that the direct influence of air upon it acts as an excitor to the respiratory movements.

That the skin acts as a respiratory organ of much importance in the lower animals, is so familiar a fact that I need not dwell upon it. The following experiment, which was performed in conjunction with Dr Sharpey, places its importance in asphyxia in a strong light, and serves to show that the respiratory function of the skin is of itself sufficient, without any action of the lungs, to excite the action of the heart, and, to a certain extent, to restore the circulation.

Exp. 12.—A middle-sized frog was immersed in soda water. Immediately before immersion the respirations were 132 in a minute, and the pulsations 56. After having been twenty-two minutes in the liquid all movement ceased. It was then withdrawn, and the heart was seen to pulsate slowly through the parietes of the thorax. This soon became so feeble as no longer to be perceptible.

Five minutes after it was removed the web was examined under the microscope; the circulation was not general, indeed almost stopped, merely going on in one or two of the larger vessels.

In eight minutes the circulation was becoming more active, with occasional interruptions only.

12*th minute.*—Heart distinctly beating 22 in a minute,—circulation active in the web. On irritating the eyeball with forceps no motion followed; pinching the skin at the verge of the anus had no effect.

18*th minute.*—Heart pulsating 26 per minute.

19*th minute.*—Animal beginning to breathe again.

22*d minute.*—Heart pulsating 29 per minute. On touching the eyelid with forceps it immediately contracted. Respiration not fully established; breathes only at intervals.

25*th minute.*—Heart beating 36 per minute.

In this experiment, which I have several times repeated with the same general results, it is evident that the increase in the frequency of the heart's pulsations, after the animal was removed from the soda water, and before it breathed, can only be accounted for by the exposure of the integuments to the action of the air, by which a certain quantity of oxygen was absorbed, and that the carbonized blood becoming thus aërated, the heart's action was restored in the same way as if that change had taken place in the lungs.

That the human integument likewise exercises a respiratory action, although to a minor degree, is equally well known. Cruickshank, Abernethy, and Collard de Martigny have fully proved that carbonic acid is evolved and oxygen absorbed when the skin is exposed to the air. Abernethy in particular has made many interesting observations on this subject, and states that, on introducing his hand into a glass jar filled with air, and inverted over mercury, carbonic acid was abundantly eliminated and oxygen absorbed. It being therefore, in the treatment of asphyxia, one of our principal objects to oxygenize the blood as rapidly and as quickly as possible, and as it has been proved by the experiments just related, that this may, to a certain extent, be accomplished in the lower animals, and, by Abernethy's observations, that it also happens in man when the integument is exposed to the air; we are, I think, from analogy as well as from actual observation, justified in concluding that, in the human subject, a certain quantity of the carbonic acid that is accumulated in the system might be eliminated, and a corresponding quantity of the asphyxial blood oxygenized by the free exposure of the integument to the vivifying influence of the atmosphere. In this way the blood in the capillaries of the whole of the surface would undergo its proper chemical changes, and would be more readily influenced by the contractions of the heart, in consequence of the removal, to a certain extent, of the obstruction to the passage of the carbonized blood that has been shown to exist in the systemic vessels. The aëration of the blood in the superficial vessels might also, perhaps, in some degree, of itself, tend to the re-establishment of the natural temperature of the body.

The direct action, likewise, of the air upon a large extent of the cutaneous surface will further, as is well known, have a tendency to excite the respiratory movements, and will thus aid our endeavours to establish the act of respiration in a natural way.

The apparatus necessary for applying hot air is exceedingly simple. The patient, after being properly dried, should be laid on the kind of bed, if it be at hand, that was used for cholera patients, and that is at present occasionally, I believe, employed at the Receiving-House of the Humane Society. It is furnished with a tin mattrass for receiving hot water. This mode of applying heat has the additional advantage over the hot bath, of the temperature being gradually and progressively increased by it, whereas the converse is the case in a bath; which is at its highest temperature at the moment that the body is immersed, and rapidly cools afterwards. If these means could not readily be put in practice, the temperature of the room in which the patient lies might be raised to 70 or 80 degrees, or the body might be exposed to the influence of a large fire, at some distance however, and a warming-pan covered with flannel might be passed repeatedly over it. Artificial respiration and other means that it might be thought advisable to employ could also be more conveniently applied to a patient lying in a bed than to an inanimate body supported in a bath. These advantages, however, great as they indubitably are, I look upon as secondary in comparison to the very important one of the exposure of so large a mass of blood as that which is contained in the capillaries of so vascular an organ as the skin to the influence of an oxydizing agent.

Whilst heat is being applied in one or other of the ways above mentioned, friction, as recommended by the Society, should be employed, either with the hand or with a horse-hair glove, or a soft flesh brush, so as to equalize the circulation as rapidly as possible, and to stimulate the action of the cutaneous vessels.

We will now proceed to make a few remarks on by far the most interesting and important question connected with the treatment of asphyxia, namely, the value of artificial respiration, and the best mode of employing it; on both of which points it would appear that considerable diversity of opinion exists amongst physiologists and practical men. Haller, Hunter, Goodwyn, Coleman, Bichat, Kay, Carpenter, and indeed almost every writer on asphyxia, from the time that the chemical changes that take place between the blood and the air in the lungs began to be understood, down to the present day, have insisted on the paramount importance of supplying the blood in the pulmonary vessels with fresh air, in some way or another, as rapidly as possible. On the other hand, the Royal Humane Society, acting under the guidance of the medical members of its committee, namely, Sir B. Brodie, Mr Dalrymple and Mr Woolley, gentlemen whose names entitle any opinion that may emanate from them to the most serious consideration of the profession, has somewhat discountenanced the employment of this means, which, I understand, is but rarely had recourse to at the institution in Hyde Park.

In that Society's report for the year 1840 we find the following paragraph. "It is the opinion of the medical members of the committee, that the period in cases of asphyxia when artificial respiration might be successful, is very short and scarcely more than momentary, and, as it but rarely happens that such measures can be applied at the precise moment, artificial respiration should be considered a secondary means." Again, in the report for the year 1842, Sir B. Brodie says, "It appears to me that there are very few of these cases in which this method is really applicable. In an individual who dies at once, either from drowning or from strangulation, the action of the heart continues during a very short period (perhaps not more than two or three minutes) after the muscles of respiration have become incapable of performing their office. If in this interval the lungs be inflated, the action of the heart will be maintained until that of the respiratory muscles is restored, and thus the life of the patient may be preserved." This most distinguished surgeon and physiologist then goes on to remark on the lapse of time that must ensue before the proper apparatus can be adjusted, and says, "We must admit that it can scarcely ever happen that life will be preserved by these means at this period when it could have been preserved otherwise.' He, however, recommends the adoption of artificial respiration in those cases of secondary asphyxia in which the patient, after having been revived for a time, gradually appears to sink again into a state of insensibility, being, as Dr Davy expresses it, "poisoned by his own blood." Sir B. Brodie is further of opinion, that the action of the heart cannot be re-excited by inflation of the lungs after it has once ceased.*

In this divided state of opinion between men whose views are equally entitled to our consideration, it will be well, in order to come to something like a conclusion upon this important subject, to examine it in detail, and more particularly with regard to the following questions:—

1st, Can artificial respiration re-establish the circulation through the lungs after it has entirely ceased?

2d, Can it re-excite the contractions of the heart after they have entirely ceased?

That insufflation of the lungs with atmospheric air can re-establish the circulation in these organs after it has entirely ceased, and after they have become congested with asphyxial blood, there can be no doubt from the following experiment, which is one

* Since these sheets were in the press I have been informed by Mr Woolley that the practice adopted by him at the Receiving House of the Society, is to employ artificial respiration in all those cases in which the respiratory actions are not naturally restored on taking the sufferer out of the water, or on placing him in the hot-bath. My observations in the text apply, therefore, solely to the published reports of the Society, and not to the practice of my friend Mr Woolley.

chosen almost at random from a considerable number on the same subject, performed by Dr Sharpey and myself.

Exp. 13.—A pipe, with a stop-cock adapted to it, was introduced into the trachea of a young middle-sized mongrel terrier; the cock was then shut.

In half a minute the animal began to struggle. The struggles continued with occasional intermission until

2½ minutes, when they ceased. At the 7th minute, the animal being quite dead, the heart was exposed and the pericardium opened; it was acting freely. Vessels on its surface were uniformly black; the arteries and veins equally so. Both the auricles were black; the right auricle was distended, the left full.

8*th minute.*—Ventricles ceased to contract regularly, but tremulous movements continued in their muscular fibres. Auricles acting strongly and regularly, from 50 to 60 in a minute. Sometimes more rapidly, about 80. Right side of heart much distended. Pulmonary artery appeared very tense. Pulmonary veins at the roots of the lungs were full and perfectly black. Lungs collapsed, mottled darkly.

13*th minute.*—Contractions of auricles less powerful. Blood still flowed from mammary arteries on being cut.

20*th.*—Auricles acting feebly, contractions chiefly, if not entirely, at the tip.

29*th.*—Auricles ceased.

30*th.*—Auricles recommenced acting, but very irregularly.

34*th.*—Auricles ceased acting for about half a minute, then began again to pulsate, then ceased again; then began again to contract about once every quarter of a minute.

37*th.*—Left auricle ceased entirely.

44*th.*—Right auricle ceased entirely; both auricles perfectly black; left auricle, to all appearance, nearly empty; right auricle distended.

45½ minutes after trachea had been closed.—Artificial respiration was now commenced.

49½ *minutes.*—Left auricle has decidedly become of a much more florid hue; on puncturing its tip, red blood escaped in small quantity; on squeezing auricle a quantity of blood of a darker hue, but not decidedly venous, escaped.

54*th minute.*—The blood that now escapes is perfectly florid; the tip of the auricle was now tied up.

57*th minute.*—Coronary arteries of a distinctly lighter colour than the veins; the femoral artery which was exposed was quite black; the branches of the pulmonary veins at the roots of the lungs of a very decidedly lighter colour than those of the pulmonary artery in the same situation, which are still black.

73*d minute.*—On puncturing the left auricle at the point of

junction with the pulmonary veins, a very large quantity of perfectly bright arterial blood issued in a small jet a line or two in height. The insufflation being continued, perfectly florid blood continued to well out each time the lung was distended.

On opening the pulmonary artery, the branches of which were very much less full than they had been, some black blood was found in it; this, however, seemed barely sufficient to fill the vessel, and certainly there was no tension of the coats of the artery. The lungs, which had been of a dark mottled hue, had become quite light coloured.

This experiment, which I have repeated a great number of times with the same results, presents several points of interest. In the first place, it clearly proves the possibility of the re-establishment of the circulation through the lungs after the heart's action has entirely ceased. For although artificial respiration was not established until 37½ minutes after the ventricles had ceased to contract, yet the blood that had stagnated in the vessels of the lungs rapidly became oxygenized, and passed in large quantity into the left cavities of the heart, as was proved not only by the free gush of arterial blood when the left auricle was punctured, but also by the pulmonary artery, which had previously been much distended, becoming lax and emptying itself almost entirely of blood.

The mechanism of the passage of the blood through the pulmonary vessels in these experiments, after the heart's action had entirely ceased, is an interesting subject. As it has already been explained in a former portion of this paper, it will not be necessary to dwell particularly upon it here. I may, however, state that it appears to be owing to the *vis-a-tergo* influence of the tense pulmonary artery forcing the blood through the pulmonary veins, after the obstacle that exists in them has been removed by the aëration of the blood contained in them. That the passage of the newly oxygenized blood into the left auricle is also materially aided by the respiratory movements, is evident from the fact of its welling out in a fuller stream from a punctured pulmonary vein, each time the lungs are distended with air. This fact, which was noticed in the experiment, the details of which have just been given, I have frequently observed, and it illustrates in a striking manner the influence of the respiratory movements on the circulation through the lungs.

Another point of interest is the length of time after which artificial respiration will still oxygenize the blood that has stagnated in the lungs. In the experiment just related, blood continued to be arterialized seventy-three minutes after the trachea had been closed, and more than an hour after the ventricles had ceased acting, and the circulation consequently been brought to a stand. I have not made any experiments to ascertain the maximum of the

time at which artificial respiration will oxygenize the blood in the pulmonary vessels, but have no doubt that it is considerably more than has even just been mentioned, probably until coagulation takes place.

The next question is, how long, after the entry of air into the lungs has been prevented, can artificial respiration restore the heart's action?

The experiments of Dr Roesler* afford very interesting evidence on this point. This gentleman found in three of his experiments that animals, (two rabbits and a cat), which had been kept under water for $5\frac{3}{4}$, $9\frac{1}{2}$, and $11\frac{3}{4}$ minutes respectively, were restored by artificial respiration, although it was not commenced until six minutes after they had been removed from the water; which time was consumed in drying aud warming the animal. In another series of eight experiments, in which the inflation was performed with warm air, and in which the duration of submersion, after life had apparently ceased, was respeetively 0, 2, 4, 4, 4, 6, 8, 8 minutes; and the whole duration of the asphyxia 7′ 50″, 9′ 50″, 11′ 50″, 11′ 50″, 11′ 50″, 13′ 50, 15′ 50″, 15′ 50″; five were saved, namely, the first, second, third, fourth, and sixth.† "The temperature of the air of inflation varied from 95° to 98° 5′; and the warmest air appears to have been most useful. The duration of the treatment was considerably shorter than was required with temperate air. It was seldom requisite to continue the treatment above 20 or 25 minutes, and once only about three-quarters of an hour. It would, however, appear that in some cases he failed although the same curative means were employed; and that even when the animals were taken out of the water as soon as they had expired. The reason of this will by and by appear.

These experiments of Dr Roesler are exceedingly valuable, as proving the length of time after which life may be restored in an animal apparently dead from drowning, and which has been submersed for a considerable period after life appeared to be extinct. For it must be remembered that, in all the cases, six minutes were consumed in rubbing and drying the animal after it had been withdrawn from the water, and before inflation was commenced. As the reviewer of Dr Roesler's experiments in the Edinburgh Medical and Surgical Journal very justly remarks, "It may be said that during the six minutes the animal lay in the air before the lungs were inflated, an obscure respiration might have been going on. But every one who is practically familiar with the phenomena of suspended animation, must know that this was impossible. After an animal has been asphyxiated so long that its breathing has ceased, the first efforts to renew respiration are

* Edin. Med. and Surg. Journal, Vol. xxiii. † Loc. cit. p. 213.

never obscure; they are convulsive gasps attended with spasm of the whole body, such as no one could possibly fail to notice, who was occupied with preparations to resuscitate it. But even granting that the animals did breathe during this period, still Dr Roesler was enabled to resuscitate, by artificial respiration, animals that had been submersed for $11\frac{3}{4}$ and $13\frac{1}{2}$ minutes."

Now, as I have already stated in a former part of this paper, the average time that I have found the ventricles to continue to contract in adult dogs is $9\frac{1}{2}$ minutes, and the longest that I have observed has been 14 minutes. Thus, then, in Dr Roesler's experiments it is very possible that, in the cases of recovery, although the animal had been immersed in water for 11 or 13 minutes, the ventricles had not entirely ceased to act before inflation was commenced; which is by no means unlikely if the animal be young. In this way we might account for the occasional failure of the same means, even when put in practice at a much earlier period, as I have occasionally found the ventricles cease to act as early as the sixth minute after the commencement of asphyxia. I believe that the principal cause of the difference in the length of time that the heart continues to contract in cases of asphyxia depends, *cæteris paribus*, upon the age of the animal. It is certainly the result of my observations, that the younger the animal is, the longer does the heart continue to contract after asphyxia has been set up. That other causes, such as the quantity of air contained in the lungs at the moment when the windpipe is closed, &c., may also have some influence is possible, but the principal one is undoubtedly the age of the animal, the irritability of muscles continuing much longer in young than in old animals.

Dr Roesler's experiments, therefore, interesting and important as they undoubtedly are, cannot fairly be adduced in confirmation of the opinion, that artificial respiration can restore the heart's action after this has entirely ceased; as the success that he had in some cases may very probably have been the consequence of artificial respiration having been set up before the ventricles had ceased to contract. The only way in which the influence of artificial respiration in restoring the heart's contractions, after these have entirely ceased, can be accurately determined, is, after an animal has been asphyxiated, to lay open the chest and expose the heart before this organ has ceased to act; and then, when its contractions have ceased, to begin the inflation of the lungs, and to note the effect produced. This I have frequently done at least a dozen times, and I can most distinctly state, that I have never in any one instance been able, by the inflation of atmospheric air, to restore the contractions of the ventricles, if they had once fairly ceased, notwithstanding that the blood in the pulmonary veins and the left auricle became florid and

arterial in the course of a few minutes. I have, indeed, occasionally noticed slight convulsive twitchings and tremulous motions in the ventricles, but never anything like regular contractile movements. What is very remarkable, however, is that very commonly the right auricle, and sometimes the left one, begins to contract regularly and forcibly after artificial respiration has been kept up for some length of time. The following experiment may be selected from amongst many others, as affording a good illustration of the phenomena of artificial respiration after the heart's action has ceased.

Exp. 14.—A small, rather young, black terrier was strangled by introducing a pipe with a stop-cock into the trachea, and then closing the cock. Strong movements of chest and fore legs ensued. In one and a half minute it was quiet.

3d minute.—Chest opened; lungs were found distended and not materially congested; blood from a cut artery perfectly black; colour of coronary arteries and of pulmonary veins quite black; left auricle dark, the same as the right.

14*th.*—Both auricles and ventricles acting feebly and at long intervals.

18*th.*—Slight movement of right auricle. The rest of heart quiet.

19*th.*—Still slight twitches of right auricle.

22*d.*—Right auricle quiet. Artificial inflation begun.

24*th.*—Pericardium opened; heart quite quiet.

36½.—Right auricle contracting distinctly and regularly, also feeble contractions of left auricle.

37*th.*—Left auricle contracts quite distinctly.

39*th.*—Contractions of left auricle more forcible and frequent.

42*d.*—Tremulous motions of right ventricle.

47*th.*—Tremulous motions of right ventricle, and the contractions of left auricle continue; right auricle quiet.

The larger branches of the pulmonary veins and artery have now been for some time nearly empty of blood, though distended before the inflation was commenced.

51*st.*—Colour of left auricle has for some time been gradually turning brighter. It is now most decidedly much brighter than that of right auricle, which has preserved its original colour. The pulmonary veins appear to contain some blood, which is of a decidedly brighter colour than what they contained before inflation. One of the branches of the coronary artery was now punctured, and perfectly florid blood escaped, and continued to drain from the opening. The accompanying coronary vein was now punctured, and was found to contain black blood.

64*th.*—Inflation stopped. Both auricles still acting, with frequent tremulous contractile movements in the right ventricle, especially about its base.

The left auricle was found to contain a moderate quantity of fluid blood, very distinctly florid. A considerable quantity drained from the pulmonary veins of a beautiful arterial tint. There were two or three small coagula in the left auricle. Left ventricle contained a considerable quantity of dark coagula, mixed with florid blood. Right cavities contained dark coagulated blood in larger quantity than the corresponding left ones. The pulmonary artery yielded a small quantity of fluid blood, lighter than that of right ventricle, but not so bright as that of pulmonary veins.

This experiment presents several points of interest; amongst others, it shows how easily arterial blood can pass into the coronary arteries independently of any contraction of the left ventricle; and that the contractions of the auricles may be restored after they have entirely ceased, although those of the ventricles cannot. In a considerable number of experiments that I have performed on this subject, I have never succeeded in re-exciting the contractions of the ventricles by means of the inflation of the lungs with common air, provided they had fairly ceased to act before artificial respiration was set up; the reason of which probably is, that when common atmospheric air is used, so long a time elapses before perfectly arterialized blood can find its way into the left cavities of the heart, that the irritability of this organ is lost to such an extent that it is no longer excitable by the stimulus of red blood. I think, therefore, that we may agree with Sir Benjamin Brodie, that the action of the heart cannot in general be restored by this means after it has once ceased. But on the other hand, there is never any difficulty in re-exciting the action of this organ, if regular contractions of the ventricles are still continuing, however feebly and slowly.

But although artificial respiration may fail in re-exciting the contractions of the heart when these have once ceased, are we on this account to discard its judicious employment in the treatment of asphyxia? I think not; for if we fail in restoring the action of the heart by a means that acts so directly upon it as that of supplying its left cavities with arterial blood does, we shall most certainly be unable to accomplish it by such measures as the application of warmth, the employment of friction, &c. which can only exert an indirect influence upon it; and which can do nothing more than maintain or equalize the circulation if it is still being carried on; but which are utterly unable to restore the contractions of the heart, or to remove the cause that has occasioned their cessation; and the less reason is there, I think, in not employing artificial respiration, as it does not, in the slightest degree, interfere with the administration of those other remedial measures that it may be thought desirable to have recourse to in the treatment of asphyxia.

The object of most of the measures employed in the recovery of persons who are asphyxiated, is necessarily to excite, in every possible way, the suspended function of respiration, and through it, the heart's action, and surely there can be no more direct way of effecting this than to employ insufflation of the lungs, which, as has been shown, rapidly unloads the congested condition of those organs and of the right side of the heart, supplying arterial blood to the left cavities, which will, if that organ be still contractile, immediately be propelled through the arterial system, stimulating the respiratory tracts of the nervous centres, and thus conducing materially to the re-establishment of normal respiration. I cannot, therefore, agree with the committee of the Humane Society in their statement, that the time during which artificial respiration can be applied with a chance of success " is very short, and scarcely more than momentary." On the contrary, it appears to me to be applicable with a likelihood of success, for at least as long a period as any of the other means recommended by the Society; that is to say, as long as the contractions of the heart continue, however feebly they may be carried on. As artificial respiration then unloads the pulmonary artery and the capillaries of the lungs, of the blood that has stagnated in them, and supplies the left side and the substance of the heart with its proper stimulus, red blood, it will, if that organ be still contractile, stimulate it more directly and rapidly than any other means that we are in possession of (for all means will fail so long as the heart, both as regards its cavities and tissue, is filled with black blood); and it cannot, I think, but be looked upon as the most important agent that we can employ in the treatment of asphyxia.

The whole value of artificial respiration, however, depends upon the manner in which it is employed. It will be needless for me to do more than to refer to the memoirs of M. Leroy upon the danger of forcible insufflation, as they are well known to all physiologists, and have already been republished in the reports of the Humane Society for 1832.

The following are the conclusions that Mr Dalrymple deduces from Leroy's experiments.

1*st*, That inflation of the lungs, when performed with haste and roughness, may be prejudicial to the safety and probable restoration of the patient.

2dly, That if the inflation, though quickly performed, be continued for some time without regularly compressing the chest for the expulsion of the air after each stroke of the bellows, accumulation of air may take place, productive of the same disastrous consequences.

3dly, That, as it is difficult to ascertain, on the first view of an asphyxial subject, in what state of distension the lungs exist, a

forced expulsion of the contained and probably frothy air should in all cases precede inflation.

Valuable as these observations are, yet they do not appear to me to militate in the least against the propriety of the employment of artificial respiration in cases of asphyxia, although they show how necessary it is to be very cautious not to use too great force. We should no more discard artificial respiration in cases of asphyxia, merely because the air-cells may be ruptured if they be unduly and forcibly distended, than we should neglect using the taxis in a case of strangulated hernia, merely because, if improper force be used, the intestines may be burst or peritonitis be excited. The dangers likely to result from the incautious use of an otherwise valuable remedy, ought not to be adduced as an argument against its guarded and judicious employment.

How, then, ought artificial respiration to be employed so as to act safely and effectually? Inflation from the mouth of an assistant is objectionable, as air once respired is not fitted for the support of animal life, and, *à fortiori*, for the resuscitation of the few sparks that may be left in the cases in which it is desirable to employ this means; and this impurity would, I think, fully counterbalance any benefit that might be derived from the increased temperature of the air so introduced,—the usual reason for which this mode of insufflation is usually recommended.

The bellows, if properly constructed so that the quantity of air injected may be measured, are no doubt very useful, and, if furnished with Leroy's trachea pipes, or what is better, with nostril-tubes, may be safely employed by medical men. But the safest, and, at the same time, a very efficient mode of introducing pure air into the lungs is either by means of the split sheet, as recommended by Leroy and Dalrymple, or else by alternately compressing the chest and abdomen with the hand, and then removing the pressure, so as to allow the thorax to expand by the natural resiliency of its parietes; and thus each time it expands to allow a certain quantity of air to be sucked into the bronchi. The quantity introduced need not be large; for, by the laws of the diffusion of gases, if fresh air be only introduced into the larger divisions of the bronchi, it will rapidly and with certainty find its way into the ultimate ramifications of these tubes. This last means of inflation has the additional advantage of resembling closely the natural process of respiration, which is one of expansion from without inwards, and not, as when the mouth or bellows are used, of pressure from within outwards. In one case, the lungs are as it were drawn outwards, the air merely rushing in to fill up the vacuum that would otherwise be produced within the thorax, by the expansion of its parietes; in the other case they are forcibly pressed upon from within, and driven outwards.

It has already been shown, that when the contractions of the

ventricles have once fairly ceased in animals, as determined by laying open the chest, they cannot, in general, be re-excited by any of the means that are in ordinary use. This is in accordance with the most trustworthy observations on the human subject. The question, therefore, necessarily presents itself, are we to leave all those persons who have been asphyxiated, and in whom the action of the heart has ceased, to their fate; and is there no possibility of devising some means by which the contractions of the heart may be re-excited and the circulation renewed? Before attempting to restore the action of the heart it is absolutely necessary to supply its left cavities with arterial blood. The reason of this is twofold. For, in the first place, as the contractions of the heart have ceased, as has already been shown, in consequence of the want of the natural stimulus to that organ—arterial blood—it is of course necessary that this stimulus be supplied before they can be re-excited; and, in the second place, even if the contractions of the heart could be re-excited the circulation could not be renewed with black blood. The only means by which the left cavities of the heart could be supplied with red blood would necessarily be by the oxygenation of that contained in the lungs, which, as has already been shown, can readily be effected even with common air. These considerations induced me to try whether the same object could not more readily and rapidly be accomplished by the introduction of pure oxygen into the lungs; and the cavities of the heart be thus rapidly filled with a highly oxygenized and very stimulating fluid.

With the view of determining this point, I have performed the following series of experiments.

I may premise that the animals had not taken food for from twenty to four-and-twenty hours before they were experimented upon; that they were all asphyxiated by closing the stop-cock of a pipe that had been introduced into the trachea; that the time dates from the commencement of the asphyxia; that the chest was laid open, but the pericardium not, and that the lungs were inflated by adapting a large bladderful of oxygen to the pipe in the animal's trachea; most generally two or even three bladdersful of gas were used to each experiment.

I. Experiments in which the heart's action was restored:—

Exp. 15. A mongrel terrier. A soon as the cock was closed the usual phenomena of asphyxia presented themselves. When the pulsations in the femoral artery had ceased to be perceptible the chest was laid open.—12*th minute.* All ventricular action has ceased, there being no tremulous movements even in the ventricles. —14*th minute.* The inflation with oxygen was commenced; it was continued for eleven minutes. At the expiration of this time the ventricles were contracting regularly and distinctly, but feebly, seventeen times in a minute. The auricles were beating about twenty-four strokes in a minute.

Exp. 16. A small mongrel terrier. In a minute and three-quarters after trachea was closed the animal was quiet, all struggles having ceased.—10*th minute.* The ventricles having now ceased all action for more than two minutes, inflation with oxygen was begun. 15*th minute.* The pulmonary veins and left auricles are of a much brighter red hue, evidently containing florid blood. The inflation was continued till the 25th minute, when it was left off. The ventricles are now acting distinctly and regularly, contracting about twenty-two times in a minute. The auricles are quite still. The contractions appear to be stronger in the right than in the left ventricles.

Exp. 17. A sheep-dog was asphyxiated. The usual phenomena ensued. At the 7*th minute* the ventricles had entirely ceased to contract. At $11\frac{1}{2}$ *minutes* the auricles were quiet, and at $12\frac{1}{2}$ *minutes* inflation with oxygen was begun. At 18*th minute* the right auricle was beating forty in a minute. At 25*th minute* the ventricles were contracting regularly and distinctly fourteen times in a minute, and on continuing the inflation their action rose to twenty-four.

Exp. 18. A mongrel spaniel. Ventricular action had entirely ceased in nine minutes. At 10*th minute* inflation with oxygen was begun. At the 20*th minute* heart was acting sixteen times in a minute, very regularly.—24*th minute.*—The ventricles were acting twenty-two times per minute.

II. In the following experiments inflation with oxygen failed to re-excite the contractions of the heart.

Exp. 19. A middle-sized old mongrel terrier. 16*th minute.*—All action of ventricles having ceased, inflation with oxygen was begun and continued for nearly twenty minutes without effect.

Exp. 20. A middle-sized terrier. 9*th minute.* All ventricular action having ceased, inflation was set up, and continued for a quarter of an hour without effect.

Exp. 22. Small terrier. 6*th minute*, after trachea had been closed, heart acting 30 per minute, inflation commenced; animal recovered very rapidly. Tube closed again, and when heart's action had ceased for one minute, inflation recommenced, but without effect.

Exp. 22. Mongrel terrier. 13*th minute.* Heart's action had ceased. At the 14*th minute* inflation set up without any effect, although it was continued for twenty minutes.

Exp. 23. An old mongrel spaniel. Heart's action ceased in 8 minutes; inflation commenced at the 9*th minute*, and continued for 25 minutes without any effect being produced.

In all these experiments, in those in which the contractions of the ventricles were renewed, as well as in those in which they were not, the pulmonary veins and the left auricle were found filled with florid arterial blood; a greater or less quantity of which had also found its way into the left ventricle and coronary arteries;

the pulmonary artery having, also, in all these cases, lost its tension.

In these experiments, then, it was found that, not only after the contractions of the ventricles, but also, in some cases, those of the auricles, had fully and entirely ceased, the action of the heart could be restored by inflating the lungs with oxygen gas. And this, it appears to me, is a result of some importance, as regards the treatment of asphyxia. There can be little doubt but that the revival of the contractions of the heart in these cases was due to the very rapid arterialization of the blood in the lungs, and its passage into the left side and tissue of the heart before the irritability of that organ had become so far extinct that it was unable to contract on the application of its usual stimulus; and that the reason why, by the inflation of the lungs with common air, it is very difficult, if not impossible, to excite perfect contractions of the ventricles, although tremulous movements in them, and proper action of the auricles may be set up, is, that the blood does not become arterialized soon enough to reach the left ventricle, and to permeate the tissue of the heart in sufficient quantity before that organ has lost its power of contracting on the application of this stimulus. It is true, that in several instances in which inflation was set up as soon as the heart's action had ceased, the contractions of that organ could not be restored; the reason of which was not very obvious, though I believe it to be connected with the age of the animal; as I think I have noticed that the contractions of the heart are more readily re-excited in the young than in the old. The number of experiments that I have performed on this subject amounting only to nine, we cannot draw any conclusions from them as to the proportional number of the cases in which recovery is likely to take place; but nevertheless they are fully sufficient to establish the fact, that the contractions of the ventricles may, after they have entirely ceased, be re-excited by the inflation of the lungs with oxygen gas.

Judging, then, from the experiments on animals, the only data that we can safely go upon, I would earnestly impress upon the Humane Society the expediency of giving the inflation of the lungs with oxygen a trial in cases of asphyxia from submersion. I am the more earnest in this entreaty, as this gas had already been found by Dr Babington,* Mr Morgan,† and by a German chemist, to be of use in the asphyxia from the inhalation of carbonic acid; although, in the cases reported, but a very small quantity of oxygen was used, and it was inhaled from a bladder.‡

In ordinary cases of submersion, (those of the first category) in

* Medico-Chirurg. Trans. Vol. i. p. 83.

† Medical Gazette, Vol. i. p. 94.

‡ Vide Casper's Wochenschrift, No. 47, 1843, and Northern Journal of Medicine for June 1844. A young man, whilst engaged in some chemical operations, was poisoned with carbonic acid gas; the ordinary means of resuscitation failed, when, on being made to inhale 2½ quarts of oxygen gas, he rapidly recovered.

Mr. Erichsen's paper.

Fig. 2

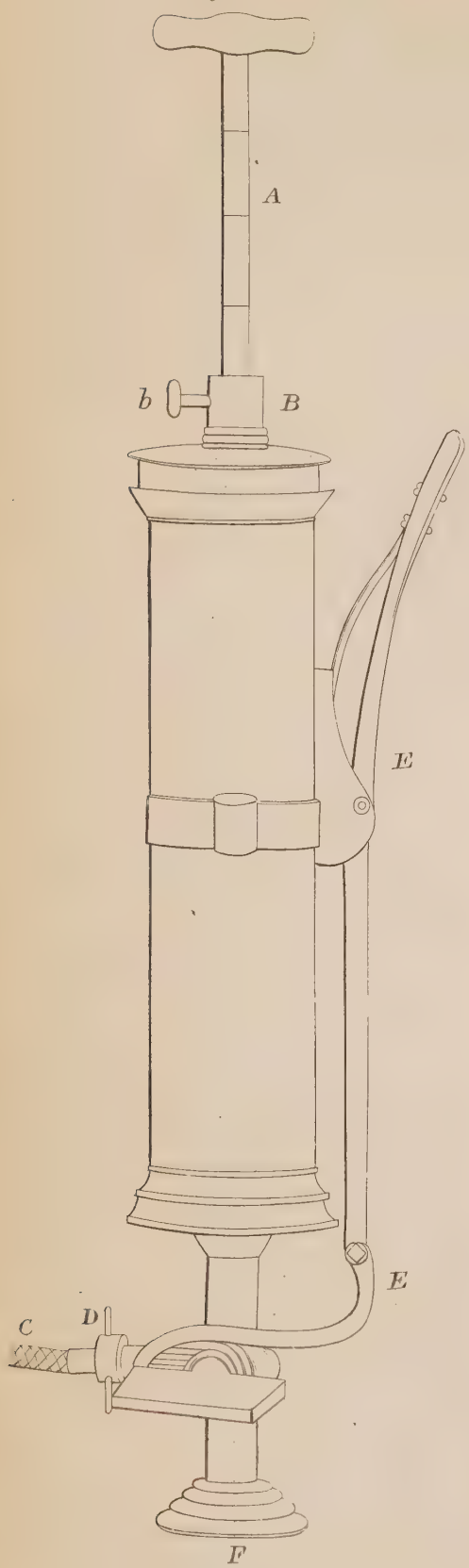
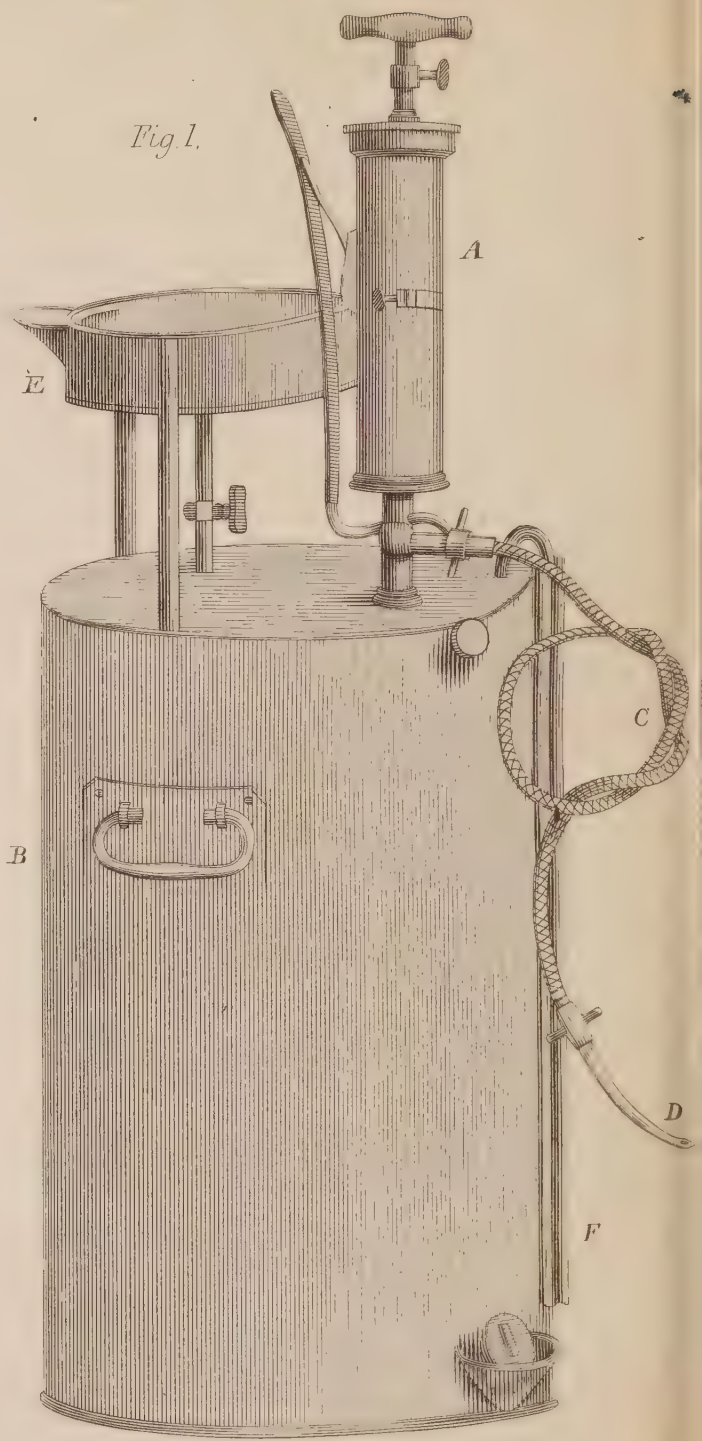


Fig. 1.



Geo. Nikman Sc.

which the means at present employed are almost uniformly successful, it would be needless, perhaps, to have recourse to this means; but in those desperate cases in which submersion has lasted for more than four minutes, or in which there is no sign of vitality left, and in which, as has already been stated, the measures at present adopted are very generally ineffectual, I would most strenuously recommend the trial of the inflation of the lungs with oxygen gas, more especially as it would not interfere with any of the means at present employed. These are desperate cases, that are now almost necessarily fatal, and that consequently justify the employment of any remedy that holds out a reasonable hope of success.

For the inflation of the lungs with oxygen, however, to be attended with a probability of success, it will be necessary to have recourse to some means by which the introduction of the gas into these organs may be effectually secured, and by which the quantity introduced may be regulated according to the circumstances of the case. A bladder or Mackintosh bag, to contain the gas, is evidently inefficient in both these respects, and has the additional disadvantage of being too small. I have accordingly contrived an instrument, of which the following is a description. This will, I think, be found to answer all the purposes required.

Description of Instrument.

Fig. 1.

A. Syringe capable of holding 20 cubic inches.
B. Gasometer capable of holding 18 gallons.
C. Elastic tube.
D. Trachea pipe, or nostril-tube.
E. Bath of gasometer.
F. Gauge of gasometer.

Fig. 2.

A. Piston-rod divided into spaces corresponding to five cubic inches of the syringe.
B. Nut sliding on piston-rod, and fixed by means of the screw *b*. The position of this nut regulates the quantity of gas injected.
C. Elastic tube ending in trachea pipe, or nostril-tube.
D. A connecting piece, by which the elastic tube is adapted to the syringe without untwisting it.
E. Flute-key valve of syringe.
F. Point of junction of syringe and gasometer.

By means of the nut that works upon the piston-rod, the quantity of oxygen injected may be correctly measured. This, I think, should not exceed fifteen cubic inches in a full-grown adult male, and must be proportionately less in women and young persons. The recent experiments of Mr Coalthupe, which appear to have been conducted with considerable care, prove that the quantity of air taken in, at an ordinary inspiration, amounts to about 22 cubic inches. If, therefore, as it is most desirable in these cases not to distend the lungs, we slowly inject about a third less than this, no danger from rupture of the air-cells need be appre-

hended, and a sufficient quantity of the gas will find its way into the air-tubes to oxygenize the blood rapidly and effectually. In consequence of the great friction exercised by the passage of a gas through a tube a yard and a half in length, the end of which is obstructed by layers of wire-gauze, the oxygen will find its way, but slowly and gently, into the lungs. And whatever be the force employed by the person who works the piston, no sudden distension of those organs can possibly be occasioned. It must be borne in mind, as has already been stated, that in performing artificial respiration there is no occasion to attempt to distend the air-cells; for if air or oxygen be introduced into the air-tubes, it will, by the well-known diffusive property of gases, rapidly find its way into the more remote divisions of those tubes, and arterialize the blood as effectually as if it had been forcibly injected.

The number of inspirations in a minute amount to about 16 or 17. In artificial inflation, however, in whatever way this is done, it is absolutely necessary to compress the chest and abdomen pretty forcibly, each time that a fresh quantity of air has been introduced, as otherwise a dangerous degree of distension of the lungs might be produced; and as it takes some little time to practise this compression effectually, it is scarcely possibly artificially to inflate the lungs more than ten or twelve times in a minute. Taking then the highest estimate, and supposing that the lungs be inflated ten times in each minute, and that at each inflation 15 cubic inches of oxygen be introduced, the quantity injected would be 150 cubic inches, or about two quarts and a half per minute, and an eighteen gallon gasometer would consequently contain enough gas to last for nearly half an hour; at the expiration of which time, the contractions of the heart would probably be restored, if it were possible to re-excite them by this means.

The apparatus that has just been described presents the additional very great recommendation, that by working it with water at a temperature of about 100°, instead of with cold water, the gas may be used warm; which, if we may judge by the analogy of Dr Roesler's experiments on insufflation with atmospheric air, increases considerably the probability of the recovery of the patient.

The chief point then, to be attended to in using this apparatus would be to use but moderate force and a moderate quantity of gas at each inflation,—to empty the lungs before beginning to inflate, and after each inflation, by compressing the chest and abdomen,—and lastly, to work the instrument with warm water.*

* There would be no difficulty in keeping a supply of oxygen at the larger receiving-houses of the Humane Society, in a zinc gasometer capable of holding 60 to 80 gallons, which might be filled when necessary at a most trifling cost; the gas prepared from the peroxide of manganese being quite pure enough for the purpose

There are several subsidiary means, such as electricity, saline injections into the veins, the inhalation of alkaline vapours, &c. the consideration of which need not detain us long.

Electric or electro-magnetic shocks passed through the heart, might perhaps, if conjoined with artificial respiration, or the inflation of the lungs with oxygen, tend to re-excite the contractions of that organ. Indeed, in two or three experiments that I have made on this subject, I have thought that they did aid in restoring the action of the heart, but my observations have not been sufficiently numerous to enable me to speak positively on the matter. I may take this opportunity of stating, that undoubtedly one of the most effective plans of treatment in cases of narcotic poisoning, when complete insensibility has supervened, is, after the stomach has been emptied of its contents, to stimulate by every means in our power the *medulla oblongata* to increased activity, so as to enable the respiratory movements to be kept up in such a way that the blood may be duly aërated; for this purpose we are in possession of no agent at all comparable to slight electric or electro-magnetic shocks passed through the brain and upper part of the spinal cord. This means, to which I believe I was the first to call the attention of the profession in the Medical Gazette for May 1841, is most easy of application, and is most effectual in exciting those portions of the nervous centres to a proper activity, on the integrity of the functions of which the persistance of the respiratory movements depends. Since I published,—three years ago, two instances of its successful application, several cases have been reported in the journals; and I have had within the last twelvemonth another opportunity of testing its efficacy in an almost hopeless case, in which an infant had been poisoned by an overdose of Dover's powder; and in which the ordinary means, such as the cold affusion, ammonia, &c. had failed in arresting the progress of the narcotism.

The injection of saline solutions into the jugular veins have been tried in two or three cases of asphyxia in dogs by Dr Sharpey and myself, but without any beneficial result ensuing. The strength of the solution used was in one case one drachm of common salt and one scruple of carbonate of soda in two pints of water; in another, the same quantity of the saline ingredients to eight ounces of fluid. I have lately heard that this means has occasionally been tried at the Humane Society's receiving-house, but without success. Indeed, from the pathology of asphyxia, it could scarcely be expected to succeed.

It has likewise been very ingeniously suggested by Dr C. J. B. Williams, that the employment of baths containing pure alkalies, or the chlorate of potass, might in some cases of asphyxia be attended with success; but I think, for reasons that have already

in question. It is needless to add that the management of the apparatus should be entrusted to none but a medical man.

been given, that more advantage would be derived from the free exposure of the surface to the vivifying influence of atmospheric air, than from the immersion of the body into any kind of bath.

It is not my intention to speak of the treatment required in other kinds of asphyxia, as in that from hanging, from the exposure to noxious gases, &c., as the directions of the Humane Society appear to me to be exceedingly judicious on these points. I may, however, state, that the very great utility of the inflation of the lungs with oxygen gas, which has already been demonstrated in the latter variety of the disease in the instances referred to, is an additional argument in favour of its employment in asphyxia from submersion.

The propriety of bleeding in cases of asphyxia depends of course upon the tendency to congestion in the head or chest after the heart's action has been fully restored, and can easily be judged of by any medical man. Care must, however, be taken not to abstract more blood than is absolutely indispensable, so that the already diminished powers of the system may not be still further exhausted by a greater loss of blood than necessary.

The danger of the supervention of secondary asphyxia after recovery has apparently taken place is now well known to all medical men, and there is, consequently, no occasion to dwell upon this part of the subject. It may, however, be stated in general terms, that the utmost care must be taken to maintain the restored actions, by keeping the patient as quiet as possible, and by attentively guarding against all causes of excitement, mental as well as bodily, that can in any way embarrass the action of the heart or respiratory organs. With this view the patient must not be allowed to walk or to make any exertion for some time after he has been apparently recovered. And if the asphyxia has been the result of an attempt at self-destruction, we must, in addition, endeavour to soothe and quiet the mind of the unfortunate sufferer.

Should symptoms of secondary asphyxia, such as stupor, laborious respiration, dilatation of the pupils, and convulsions, manifest themselves, artificial respiration should be immediately set up, and be maintained until the action of the heart has been fully restored. In these cases I should, from the very great efficacy of electricity in the somewhat similar condition resulting from the administration of the narcotic poisons, be disposed to recommend slight shocks to be passed through the base of the brain and upper portion of the spinal chord, so as to stimulate the respiratory tracts.

There are yet several points in the pathology as well as in the treatment of asphyxia, the investigation of which I had proposed to myself, but the natural repugnance that one feels to the sacrifice of animal life, however useful may be the end that is to be obtained, has induced me to relinquish my original intention.